

**“A PROSPECTIVE RANDOMIZED CONTROLLED
STUDY COMPARING THE ANALGESIC EFFICACY OF
DEXAMETHASONE WHEN ADDED TO BUPIVACAINE
IN SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK
FOR UPPER LIMB SURGERIES”**

Submitted to

**THE TAMILNADU DR.M.G.R
MEDICAL UNIVERSITY**

*in partial fulfillment of regulations
for award of the degree of*

**M.D (ANESTHESIOLOGY)
BRANCH – X**



**ESIC MEDICAL COLLEGE & PGIMSR
K.K.NAGAR, CHENNAI – 78.**

**THE TAMILNADU DR. M.G.R MEDICAL UNIVERSITY
CHENNAI, TAMILNADU**

APRIL 2015

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I solemnly declare that this dissertation titled “**A PROSPECTIVE RANDOMIZED CONTROLLED STUDY COMPARING THE ANALGESIC EFFICACY OF DEXAMETHASONE WHEN ADDED TO BUPIVACAINE IN SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK FOR UPPER LIMB SURGERIES**” has been conducted by me at ESIC Medical College & PGIMSR, Chennai, under the guidance and supervision of **Prof.Dr.Kamalini Sridharan,M.D.,D.A.,** and **Dr.K.Radhika,M.D.,** Department of Anesthesiology, ESIC Medical College & PGIMSR, Chennai. This dissertation is submitted to **The Tamil Nadu Dr. M.G.R. Medical University, Chennai** in partial fulfillment of the University regulations for the award of the degree of **M.D. Branch X (Anesthesiology).**

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Signature of the Candidate

Place: Chennai

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CERTIFICATE OF APPROVAL

To

Dr. P. Nishitha
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Dear Dr. P. Nishitha,

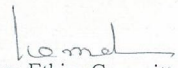
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INTRODUCTION

"Pain has been defined as an unpleasant sensory or emotional experience associated with actual or potential tissue damage".

Pain is gaining importance as the fifth vital sign along with pulse rate, blood pressure, respiratory rate and temperature. Anaesthesia for surgery should be a pleasant experiences to the patient hence the anaesthesia plan should also account for postoperative pain relief.

"Peripheral nerve block can be used as a sole anaesthetic technique. When used alone side effects of sedation, nausea and vomiting due to opioids are avoided. Patients are pain free and can be discharged as day care. Sedation when combined with nerve block improves patient comfort and tolerance of block. Brachial plexus block is a type of regional anaesthetic technique for upper limb surgeries. The plexus can be blocked at the level of the roots, trunks cords or the divisions. The block can be performed by using the ultrasound guidance or with nerve stimulator or using the land marks as a guidance. Local anaesthetics agents

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I am thankful to my parents for their blessings for their unconditional love, support and time.

Dr. P.Nishitha

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ANNEXURES

“A Prospective Randomized Controlled Study Comparing The Analgesic Efficacy Of Dexamethasone When Added To Bupivacaine In Supraclavicular Brachial Plexus Block For Upper Limb Surgeries ”

Dr.P.Nishitha, Prof.Dr.Kamalini Sridharan , Dr.K.Radhika

Background:

Different adjuvants have been used to improve the quality and increase the duration of local anesthetics during various nerve blocks. The current study compared the analgesic efficacy of dexamethasone as an adjuvant to bupivacaine in supraclavicular brachial plexus block .

Aims and objectives:

To compare the analgesic efficacy of dexamethasone added to bupivacaine and bupivacaine alone in supraclavicular brachial plexus block for upper limb surgeries on various parameters like onset of sensory and motor blockade and duration of analgesia.

Materials and methods:

80 patients undergoing upper limb surgeries under supraclavicular block were grouped into Group D (study group) who received 0.5% bupivacaine (25 ml) and epinephrine 1ml of 1 in 1000 ml + dexamethasone 8 mg (2ml) and Group B(control group) who received 0.5% bupivacaine (25 ml) and epinephrine 1ml of 1 in 1000 + distilled water 2ml.Total volume in both groups were 28ml.

Results:

Mean onset of sensory block 4.2 ± 0.99 , 13.6 ± 6 minutes in group D and group B. Mean onset of motor block 7.5 ± 3.2 , 11.5 ± 3.43 minutes in group D and group B. Duration of analgesia 18.97 ± 2.67 , 9.10 ± 1.79 hours in group D and group B. The early onset of motor and sensory block and duration of analgesia were statistically significant in group D. The time to reach vas score of 2 and 4 were significantly longer in group D when compared to group B.

Conclusion:

Dexamethasone in the dose of 8mg given as an adjuvant in supraclavicular block , results in early onset of motor and sensory block .The duration of analgesia is also prolonged significantly . This prolonged postoperative pain relief helps in early mobilisation and thereby improving physical and psychological wellbeing of the patient . This dose is optimal in that it does not cause any any side effects, at the same time it provides excellent perioperative and postoperative analgesia.

Keywords:

Supraclavicular block, bupivacaine , Dexamethasone.

INTRODUCTION

“Pain has been defined as an unpleasant sensory or emotional experience associated with actual or potential tissue damage¹”.

Pain is gaining importance as the fifth vital sign along with pulse rate, blood pressure, respiratory rate and temperature. Anaesthesia for surgery should be a pleasant experience to the patient hence the anaesthesia plan should also account for postoperative pain relief.

Peripheral nerve block can be used as a sole anaesthetic technique. When used alone side effects of sedation, nausea and vomiting due to opioids are avoided. Patients are pain free and can be discharged as day care. Sedation when combined with nerve block improves patient comfort and tolerance of block. Brachial plexus block is a type of regional anaesthetic technique for upper limb surgeries. The plexus can be blocked at the level of the roots, trunks, cords or the divisions. The block can be performed by using the ultrasound guidance or with nerve stimulator or using the land marks as a guidance. Local anaesthetic agents can be given either solely or an adjuvant drug can be added, which prolongs the duration of the plexus blockade.

The adjuvant drug may be in the form of vaso constrictor , for example epinephrine² or sedatives like tramadol³, fentanyl⁴ and buprenorphine⁵. Alpha receptor agonist like clonidine^{2,6} and dexmedetomidine⁷ have also been used safely. Steroids like dexamethasone have been found to have an effect on prolonging the block duration.

AIM

- **TO COMPARE THE ANALGESIC EFFICACY OF DEXAMETHASONE ADDED TO BUPIVACAINE VERSUS BUPIVACAINE ALONE IN SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK FOR UPPER LIMB SURGERIES.**

OBJECTIVE

- **ONSET OF SENSORY AND MOTOR BLOCKADE.**
- **DURATION OF ANALGESIA.**

REVIEW OF LITERATURE

Shrestha BR et al in another study compared the analgesic efficacy of local anesthetic with and without dexamethasone (8 mg) in supraclavicular brachial plexus block in forty patients . Addition of dexamethasone for brachial plexus block significantly prolonged the duration of analgesia without any unwanted effects⁸.

In double blinded study by Movafegh A et al, the effect of dexamethasone added to lidocaine on the onset and duration of axillary brachial plexus block was evaluated in sixty patients . 34 ml of 1.5% lidocaine was used in axillary block with or without 8 mg of dexamethasone. It was concluded that the addition of dexamethasone(8mg) to lidocaine 1.5% solution in axillary block significantly prolongs the duration of both sensory and motor blockade⁹.

Shrestha BR in a double blinded study evaluated post operative analgesia after the addition of Tramadol (2mg/kg) or dexamethasone (8 mg) to bupivacaine in supraclavicular blocks . Sixty patients was

studied. Duration of analgesia was two and half times prolonged in dexamethasone group compared to tramadol group¹⁰.

Yadav RK et al did a study comparing dexamethasone and neostigmine as adjuvants added to lignocaine with adrenaline for brachial plexus block. They concluded that the onset of blockade and the duration of analgesia was better in third (dexamethasone)group¹¹.

In a study conducted by Parrington et al , ultrasound guidance was used to administer supraclavicular brachial plexus block. 30 ml of mepivacaine 1.5% was used with or without 8 mg dexamethasone as an adjuvant. Their study concluded that the addition of dexamethasone 8 mg to mepivacaine significantly prolonged the duration of analgesia but does not reduce the onset time of sensory and motor blockade¹².

Vieira PA et al did another randomised double blinded study to determine the addition of dexamethasone to interscalene brachial plexus block in eighty eight individuals undergoing shoulder arthroscopy. 20 ml of the bupivacaine 0.5% with 1:200,000 epinephrine and clonidine 75 micrograms was used . They concluded that addition of dexamethasone to a bupivacaine epinephrine-clonidine in interscalene block prolongs sensory block and reduces opioid use¹³.

Islam SM et al did a study comparing lignocaine bupivacaine mixture of local anesthetic 35 ml with or without addition of dexamethasone 8 mg. They concluded that addition of dexamethasone as an adjuvant to local anaesthetics in brachial plexus block results in significantly early onset and markedly prolonged duration of analgesia without any unwanted effects¹⁴.

K. C. Cummings III et al in another study compared bupivacaine or ropivacaine alone or dexamethasone 8 mg added either to bupivacaine or ropivacaine in interscalene nerve blocks . Volume of the local anesthetic used was 30 ml. They concluded that when dexamethasone (8mg) is added to ropivavaine or bupivacaine , the analgesia is prolonged to 22 hours in both the group of patients¹⁵.

[Tandoc](#) et al in another study compared the effects of adding two different doses of dexamethasone on the duration and quality of interscalene block in ninety patients. One group received 4mg of dexamethasone and another group received 8 mg of dexamethasone. They concluded the addition of dexamethasone to bupivacaine significantly prolonged the duration of the motor block and improved the quality of analgesia following interscalene block. There was no difference in the duration of analgesia and motor block between low-dose and high-dose dexamethasone¹⁶.

Pathak et al in another study compared supraclavicular brachial plexus block with and without dexamethasone (8mg). 38 ml of the local anesthetic was given. They concluded that addition of dexamethasone to local anaesthetic drugs in brachial plexus block significantly prolongs the duration of analgesia and motor block in patients and is remarkably safe and cost effective method of providing post operative analgesia¹⁷.

Yaghoobi et al in another study compared the analgesic efficacy of the dexamethasone and fentanyl added to lidocaine using axillary block in seventy-eight patients. They concluded that addition of dexamethasone to lidocaine significantly prolonged the duration of analgesia compared with fentanyl/lidocaine mixture or plain lidocaine¹⁸.

In a retrospective database analysis, Rasmussen et al reviewed 1,040 patient records collected in an orthopedic outpatient surgery center that had received an upper or lower extremity peripheral nerve block with ropivacaine 0.5% with or without dexamethasone and/or epinephrine. The primary outcome evaluated was the duration of analgesia in upper or lower extremity blocks containing dexamethasone as an adjuvant. They concluded that the addition of dexamethasone to 0.5% ropivacaine prolongs the duration of peripheral nerve blocks of both the upper and lower extremity¹⁹.

Recently, Choi et al reviewed the contemporary literature and did a meta - analysis of all randomised control trials on the effects of dexamethasone as a local anaesthetic adjuvant for brachial plexus block. The objectives of meta-analysis were to assess the contemporary literature and quantify the effects of dexamethasone on Brachial plexus block. Meta - analysis was performed as nine trials in 801 patients with 393 patients receiving dexamethasone (4–10 mg).

Dexamethasone prolonged the analgesic duration for long-acting local anesthetic. The most recent trial demonstrated equivalent prolongation with perineural or systemic administration of dexamethasone compared with placebo. They concluded that perineural administration of dexamethasone with local anaesthetic prolongs brachial plexus block effects with no observed adverse events²⁰.

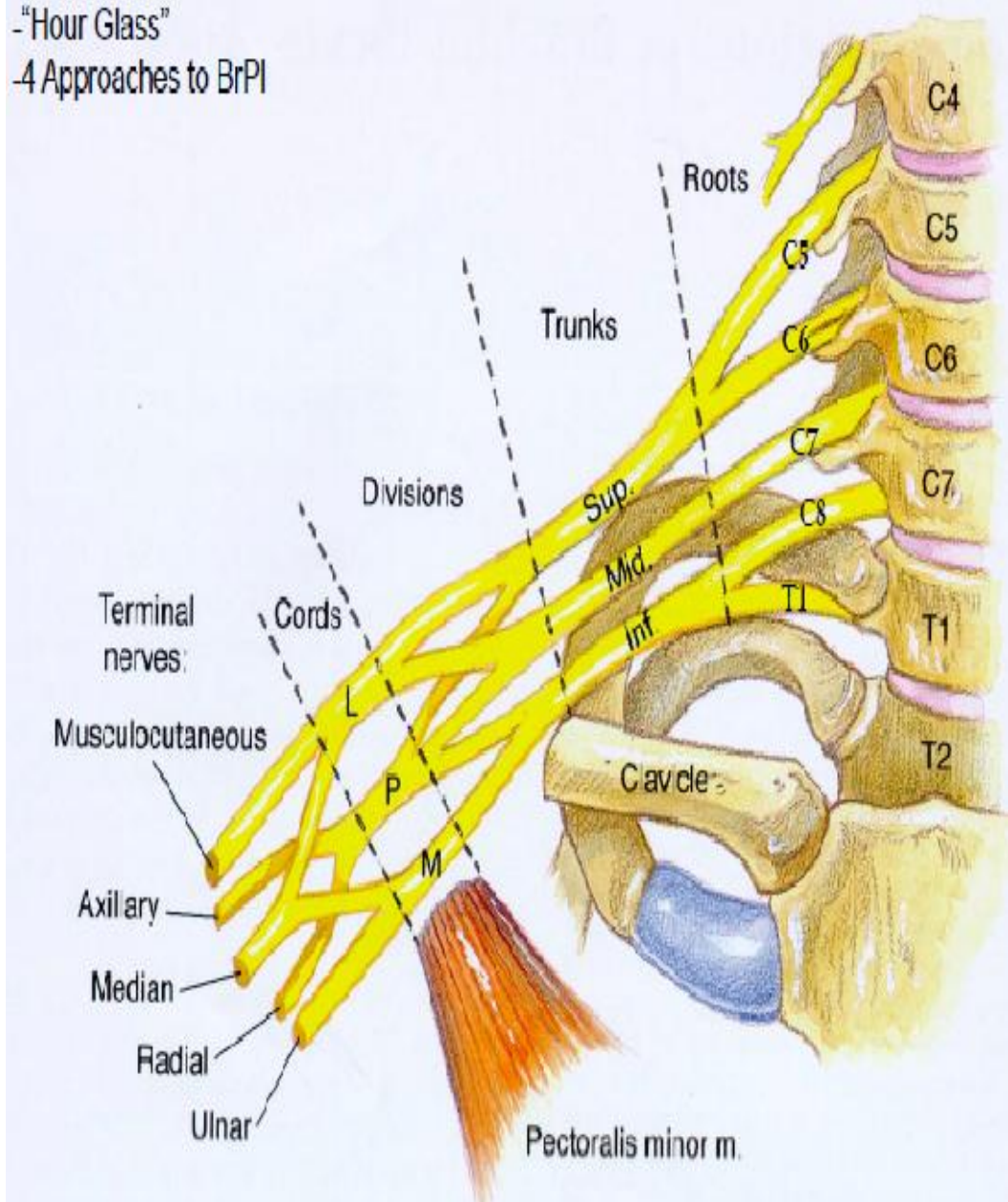
Santhosh kumar et al did a comparative evaluation of ropivacaine and ropivacaine with dexamethasone in supraclavicular brachial block for post operative analgesia in eighty patients. 40 ml of local anesthetic solution was given to both group of patients. They concluded that dexamethasone 8 mg as an adjuvant had significantly prolonged both motor and sensory blockade than ropivacaine alone²¹.

BRACHIAL PLEXUS ANATOMY

FORMATION FROM VENTRAL RAMI

The brachial plexus is formed from the ventral rami of the lower four cervical nerves and first thoracic nerves to give rise to the root value of C5, C6, C7, C8 and T1. C4 and T2 make minor contributions. These are collectively known as the ROOTS of the ventral rami. After exiting through the corresponding intervertebral foramen, the roots of the plexus are found in the cervical paravertebral space, between the anterior and middle scalene muscles. The ventral rami contains both the sensory and motor fibres supplying the anterior trunks and limbs and over lying skin²².

- "Hour Glass"
- 4 Approaches to BrPI



Interscalene block is given at the level of roots . This block is effective for shoulder and upper arm surgeries. Ulnar nerve is spared in this block.

TRUNKS : Ventral rami unite to form three trunks. The trunks are formed between the scalene muscle and the upper border of the clavicle in the posterior triangle of the neck²².

UPPER TRUNK	C5,C6
MIDDLE TRUNK	C7
LOWER TRUNK	C8-T1

Upper, middle, and lower trunks course over the lateral border of the first rib and under the clavicle. Supraclavicular block is given at the level of trunks. No nerve is spared in this block. This block is given for arm , forearm, hand surgeries.

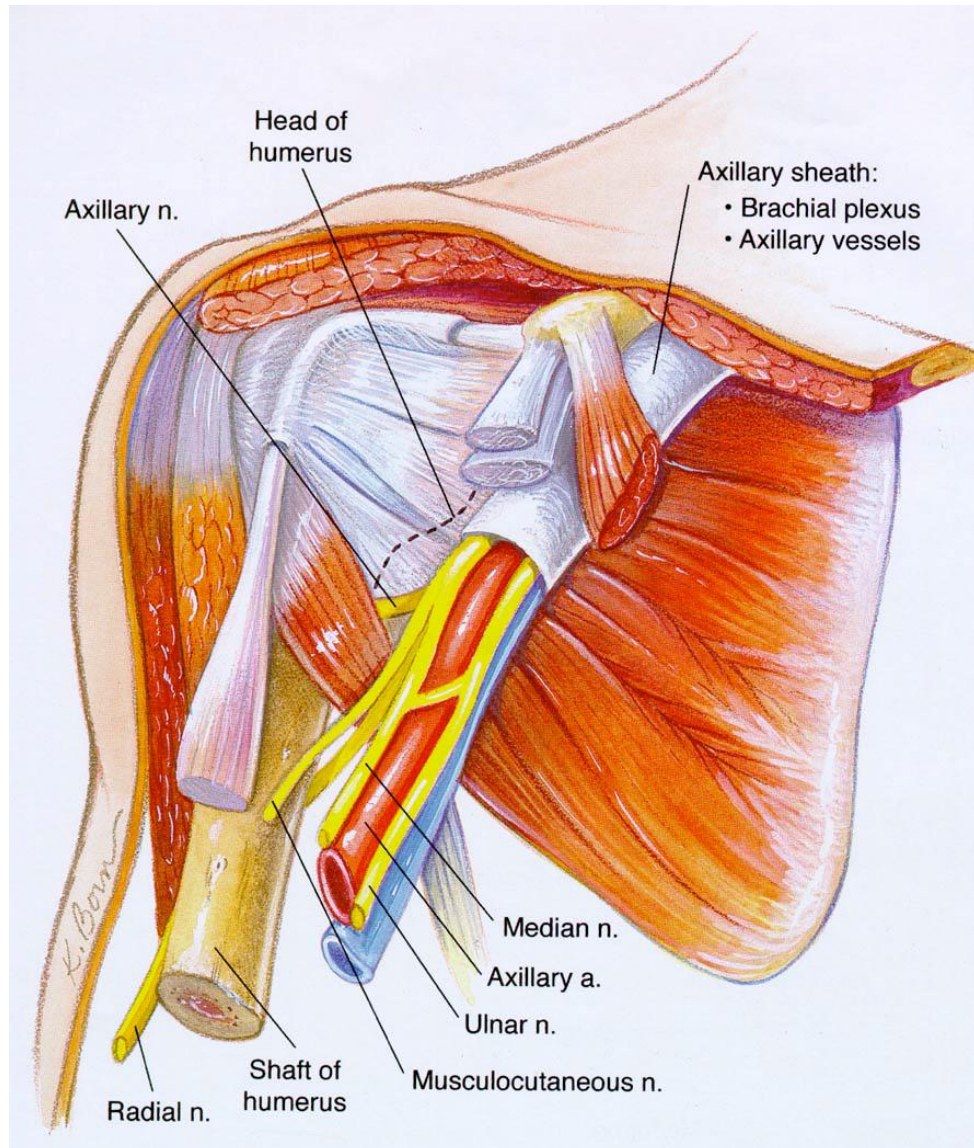
DIVISIONS

Under this location, each trunk will separate into anterior and posterior divisions which supplies extensor and flexor compartments²².

CORD

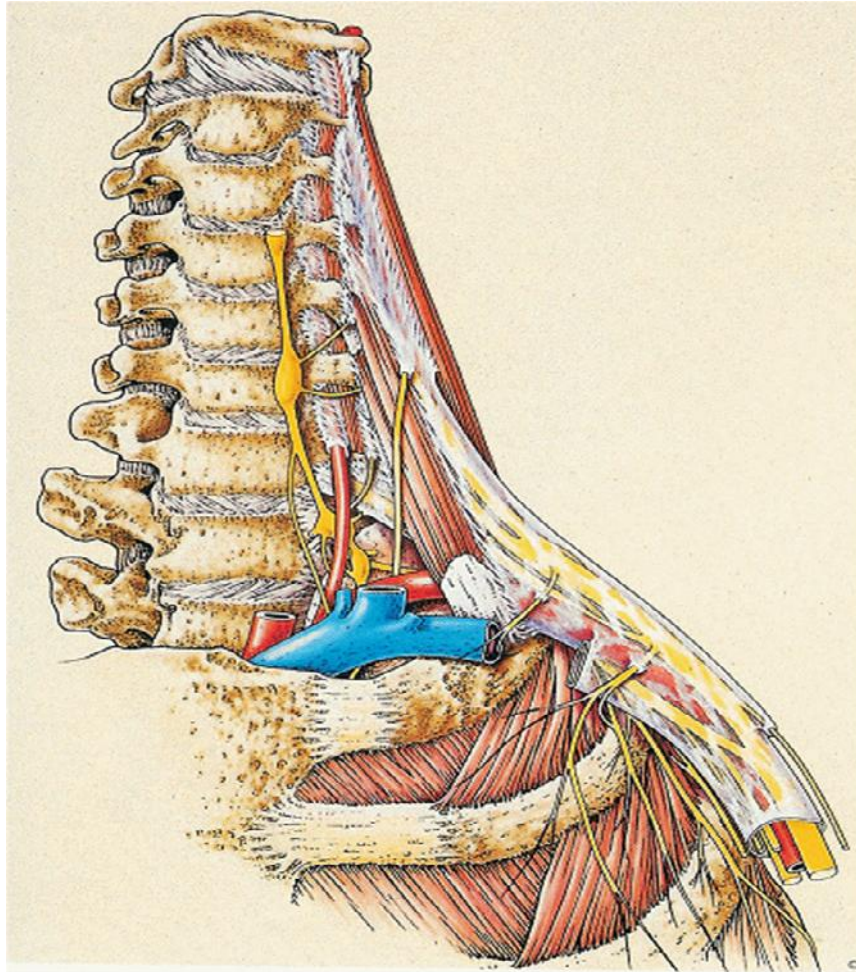
As the brachial plexus emerges under the clavicle, the anterior and posterior divisions come together to form three cords. The lateral cord is lateral to the axillary artery; the posterior cord is located posterior to the axillary artery; and the medial cord is located medial to the axillary artery²².

LATERAL CORD – formed by the anterior divisions of the upper and middle trunks.
POSTERIOR CORD - formed by the posterior divisions of all three trunks.
MEDIAL CORD - formed by the anterior divisions of the lower trunk.

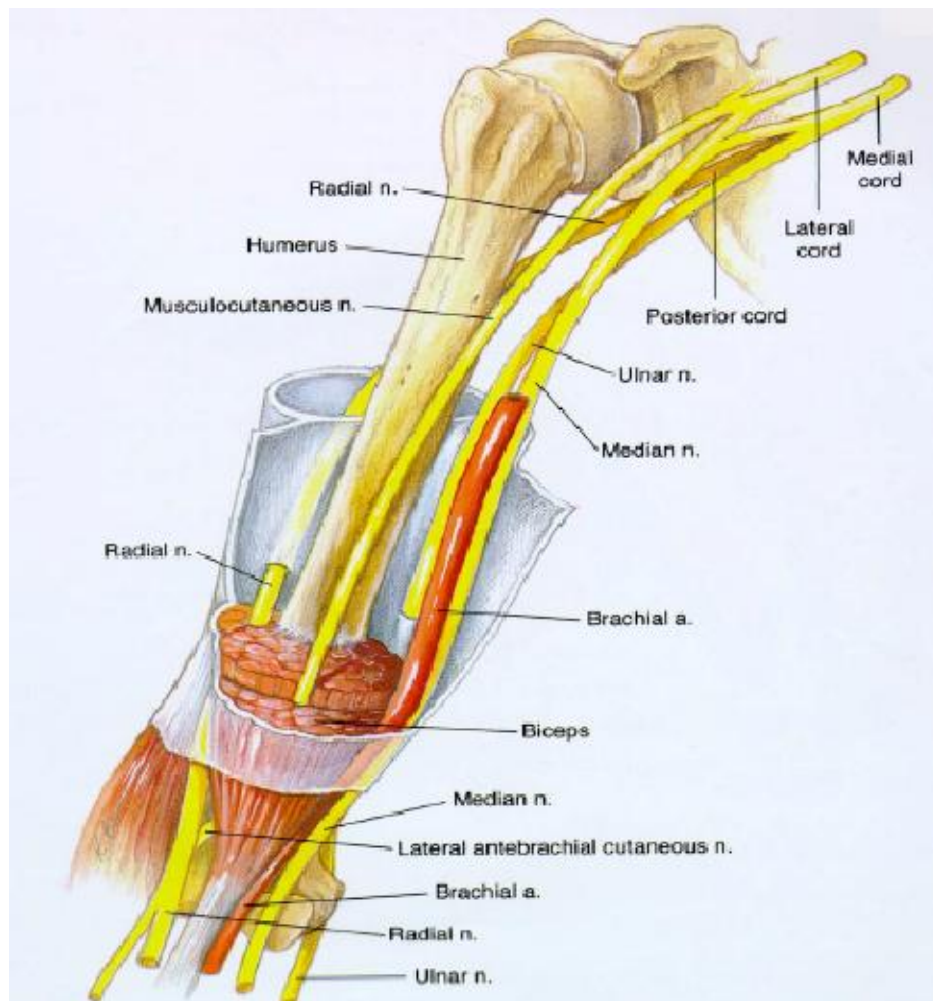


At the lateral border of the pectoralis minor muscle, each cord divides into branches, terminating in individual nerves.

Axillary block is given at the level of terminal branches. It is given for forearm and hand surgeries. Musculocutaneous nerve is spared in this block.



A sheath surrounds the brachial plexus, from the transverse processes all the way down into the axilla.



The brachial plexus is enveloped by a fascial sheath, formed by prevertebral and scalene fascia, extending from the intervertebral foramina to the upper arm. The formation of a sheath allows for the administration of brachial plexus anesthesia.

Injection into the sheath, at any anatomical point, will allow for the spread of local anesthetics and subsequent blockade. Each approach to the brachial plexus impacts specific anatomical areas of the upper extremity. Choice of a specific technique should be made based on the surgical procedure.

Distribution of the branches of the brachial plexus

Axillary nerve (C5-C6): gives an articular branch to the shoulder joint, motor innervation to the deltoid and teres minor muscles and sensory innervation to part of deltoid and scapular regions.

Radial nerve (C5-C6-C7-C8): supplies the skin of the posterior and lateral arm down to the elbow, the posterior forearm down to the wrist, lateral part of the dorsum of the hand and the dorsal surface of the first three and one-half fingers proximal to the nail beds. It also provides motor innervation to the triceps, anconeus, part of the brachialis, brachioradialis, extensor carpi radialis and all the extensor muscles of the posterior compartment of the forearm. Its injury produces a characteristic “wrist drop”.

Median nerve (C5-C6-C7-C8-T1): gives off no cutaneous or motor branches in the axilla or the arm. In the forearm it provides motor innervation to the anterior compartment except the flexor carpi ulnaris and the medial half of the flexor digitorum profundus (ulnar nerve). In the hand it provides motor innervation to the thenar eminence and the first two lumbricals. It provides the sensory innervation of the lateral half of the palm of the hand and dorsum of first three and one half fingers including the nail beds.

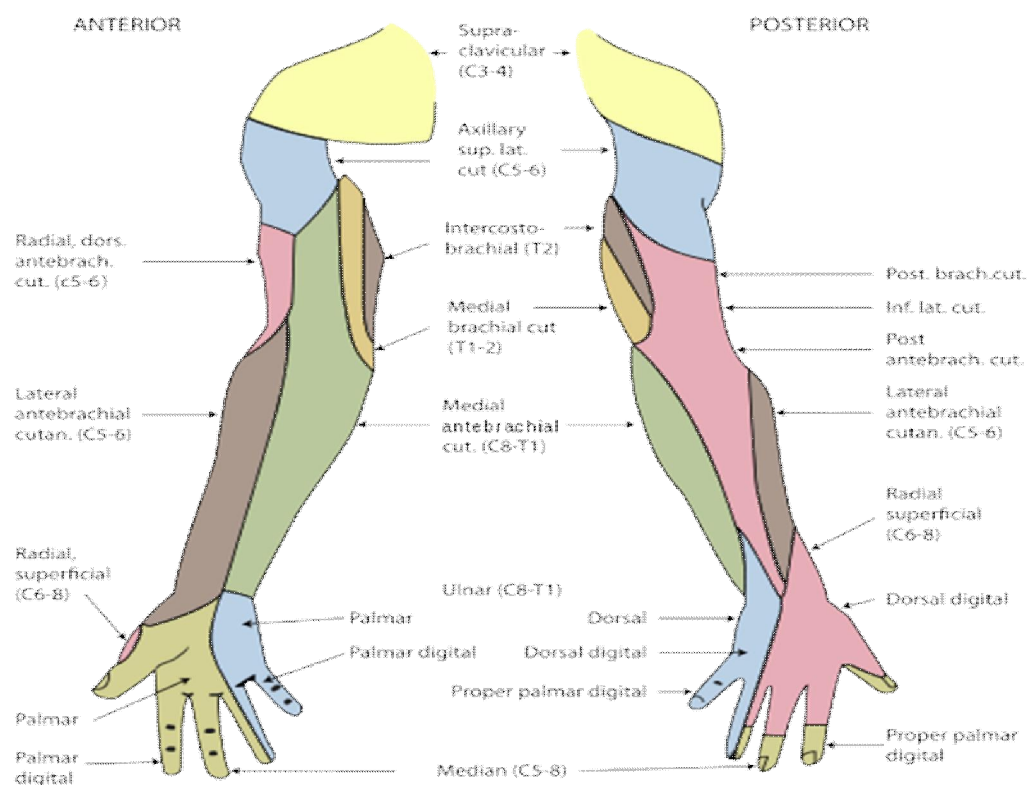
Ulnar nerve (C8-T1): like the median nerve, the ulnar nerve does not give off branches in the axilla or the arm. Its motor component supplies the flexor carpi ulnaris and the medial half of the flexor digitorum profundus. In the hand it provides the motor supply to all the small muscles of the hand except the thenar eminence and first two lumbricals (median). Its sensory branches supply the medial third of the dorsum and palmar sides of the hand and dorsum of the 5th finger and dorsum of the medial side of 4th finger.

Medial brachial cutaneous nerve (T1): it is solely a sensory nerve. It supplies the skin of the medial side of the arm. It is joined here by the intercostobrachial nerve, branch of the second intercostal.

Medial antebrachial cutaneous nerve (C8-T1): It is also a sensory nerve. It supplies the medial side of the anterior forearm.

Musculocutaneous nerve (C5-C6-C7): gives motor innervation to the coracobrachialis, biceps and brachialis muscles. At the elbow it becomes purely sensory innervating the lateral anterior aspect of the forearm to the wrist.

Cutaneous Nerve Supply of the upper limb

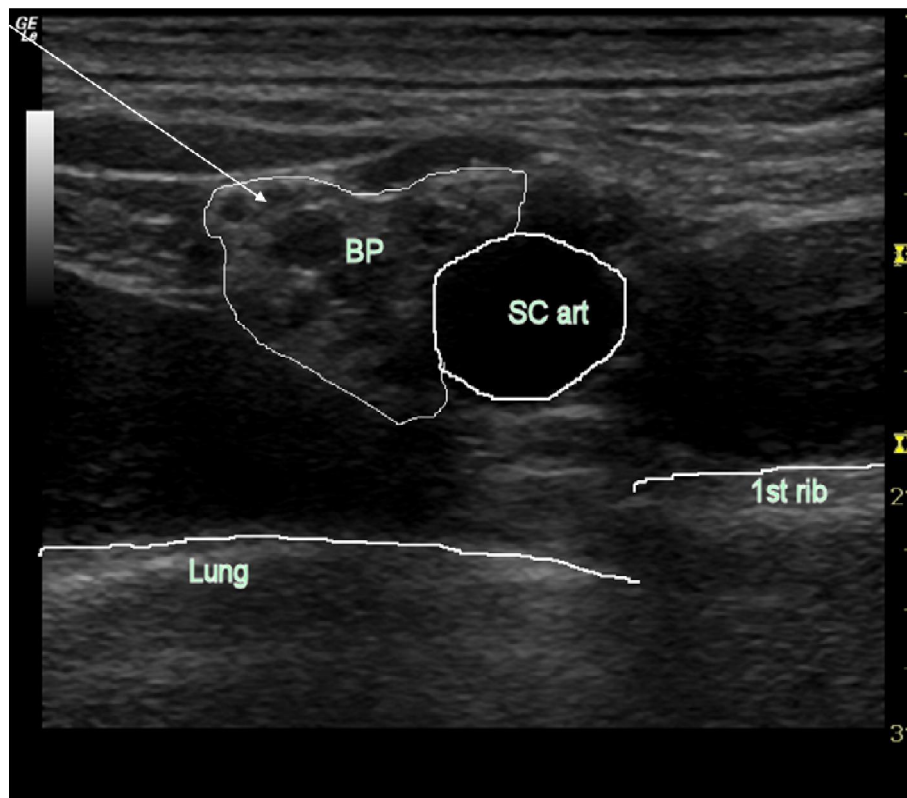


This figure shows the cutaneous innervation and root values of the terminal branches of the brachial plexus.

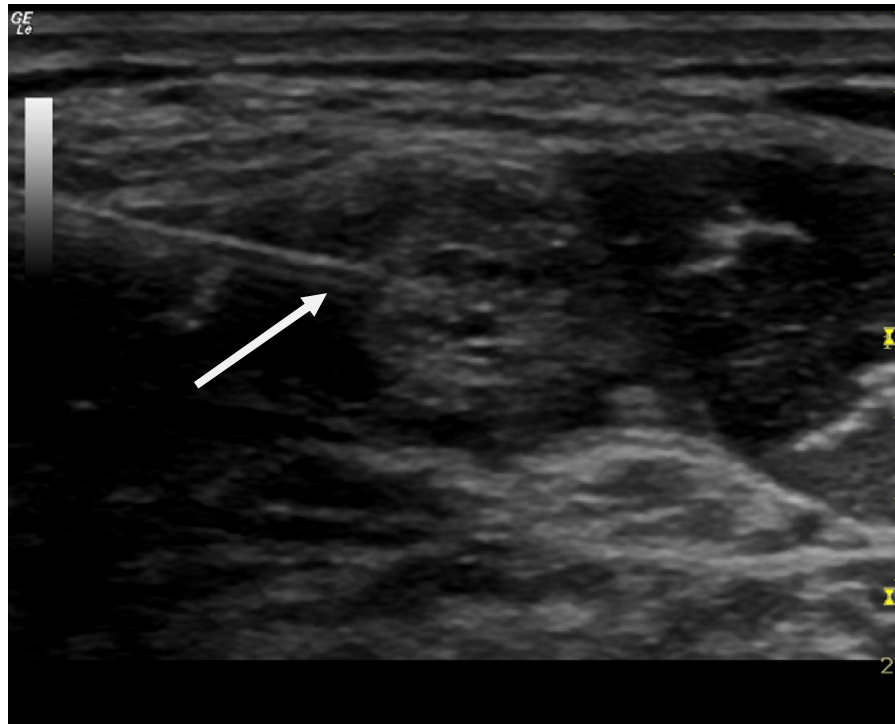
Major Motor Function of the Individual Nerves .

Nerve	Motor Function
Axillary	Abduction of the shoulder
Musculocutaneous	Flexion of the elbow
Radial	Extension of elbow, wrist, and finger
Median	Flexion of the wrist and finger
Ulnar	Flexion of the wrist and finger.

Sonoanatomy showing the brachial plexus between the scalene muscle



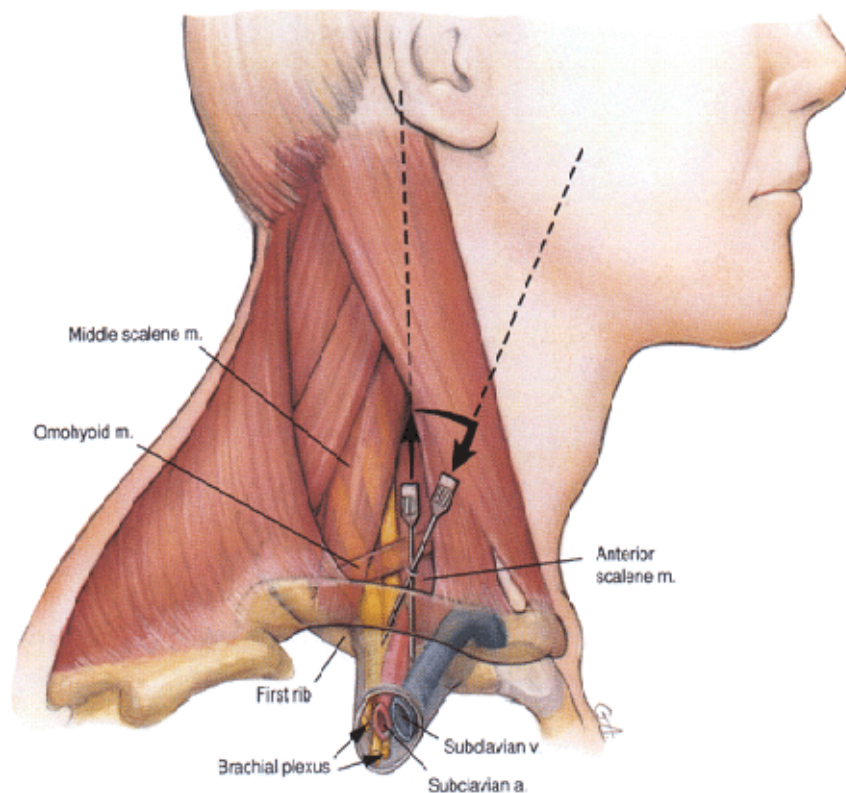
This figure shows the sonographic relationship of the brachial plexus in relation to the subclavian artery, the first rib and the pleura .



This figure shows the sonographic image of the spread of the local anesthetic within the brachial plexus

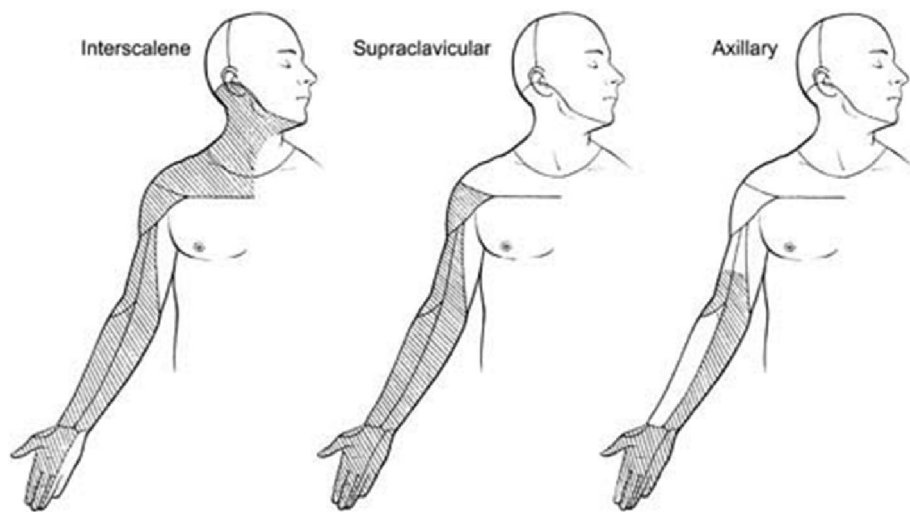
BRACHIAL PLEXUS BLOCK

First performed by Halsted in 1841. It blocks the trunks roots, divisions and cords of the brachial plexus. Its anatomic compactness is responsible for complete and reliable anaesthesia. Advantage include rapid onset and ability to perform with the patient's arm in any position. It involves injecting the local anesthetic drug in the fascial planes surrounding the nerve plexus and thereby blocking the autonomic, motor and sensory fibers supplying the upper extremity.²²



ANATOMICAL TECHNIQUES OF BLOCKADE OF UPPER EXTREMITY

- Inter-scalene brachial plexus block
- Supraclavicular brachial plexus block
- Vertical infraclavicular plexus block
- Suprascapular nerve block
- Axillary plexus block
- Blocks in the upper arm region (mid-humeral approach for the radial nerve)
- Blocks in the region of the elbow (radial, musculocutaneous, median and ulnar nerves).

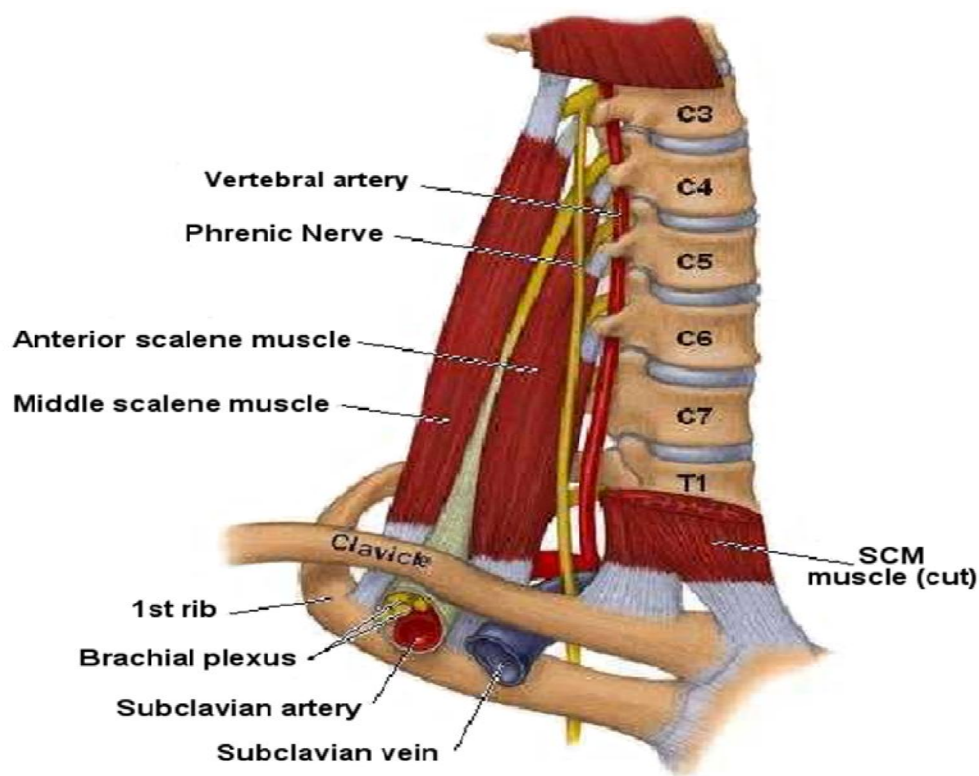


SUPRACLAVICULAR BLOCK :(Blocks the dermatome C5 to T1)

- The supraclavicular block is often called as "spinal anesthesia of the upper extremity" because of its ubiquitous application for upper extremity surgery. The first percutaneous supraclavicular block was performed by Kulenkampff in Germany in 1911. A few months after, Hirschel described a method of brachial plexus with an axillary approach. In 1928, Kulenkampff and Persky published their experiences with a thousand blocks without apparent major complications.
- The block is performed at the level of the distal trunks and origin of the divisions, where the brachial plexus is confined to its smallest

surface area . The three trunks carry the entire sensory, motor, and sympathetic innervation of the upper extremity, with the exception of the uppermost part of the medial side of the arm (T2).

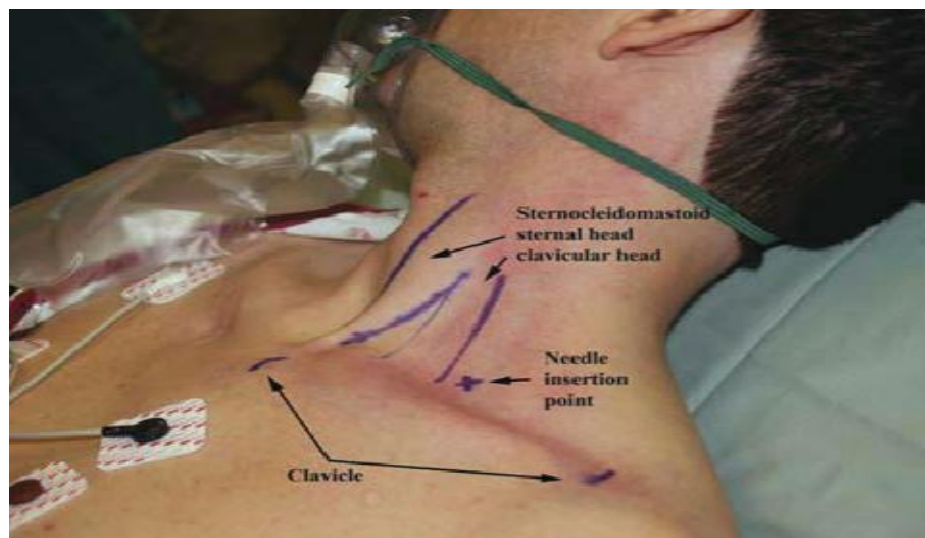
RELATIONSHIP OF BRACHIAL PLEXUS



Brachial plexus is contained within a fascial sheath. Subclavian artery lies medial to plexus as they cross the 1st rib together. Note location of phrenic nerve and vertebral artery.

LANDMARKS

Another important anatomic feature of the supraclavicular block is the presence of the subclavian artery in front of the lower trunk and its divisions . To increase the chance of blocking C8-T1 dermatomes it may be beneficial to insert the needle in the proximity of the lower trunk and make it the focal point of injection.



The sternocleidomastoid muscle inserts on the medial third of the clavicle, the trapezius inserts on the lateral third, and the neurovascular bundle passes underneath the middle third, which includes the midpoint of the clavicle. During a supraclavicular block, the pleura potentially can be breached either at the pleural dome (more likely) or through the first intercostal space. A practical knowledge of the anatomical position of the pleura is important to decrease the risk of pneumothorax. The pleural

dome is contained within the concavity of the first rib. Because the first rib crosses under the junction between the medial and middle thirds of the clavicle its path coincides with the insertion of the sternocleidomastoid muscle, which inserts on the medial third of the clavicle. Therefore, the lateral insertion of the sternocleidomastoid muscle on the clavicle can be used as a landmark for the location of the first rib and of the lateral edge of the dome of the pleura.

INDICATIONS

Surgeries involving the entire upper extremity distal to the shoulder, including the upper arm and elbow as well as the forearm, wrist, and hand.

APPROACHES TO SUPRACLAVICULAR BLOCK

1. The classical approach of Kulenkampff.
2. The subclavian perivascular approach of Winnie and Collins.
3. The modified lateral paravascular approach of Moorthy.

For supraclavicular block , the patient should be positioned supine and neck rotated slightly to the contralateral side.

CLASSICAL APPROACH

In the midclavicular line, 1cm to the posterior to the clavicle and lateral to the subclavian artery, the needle is placed posteriorly, medially and caudally until the brachial plexus is located. If not found the needle is walked along the clavicle.

COMPLICATIONS

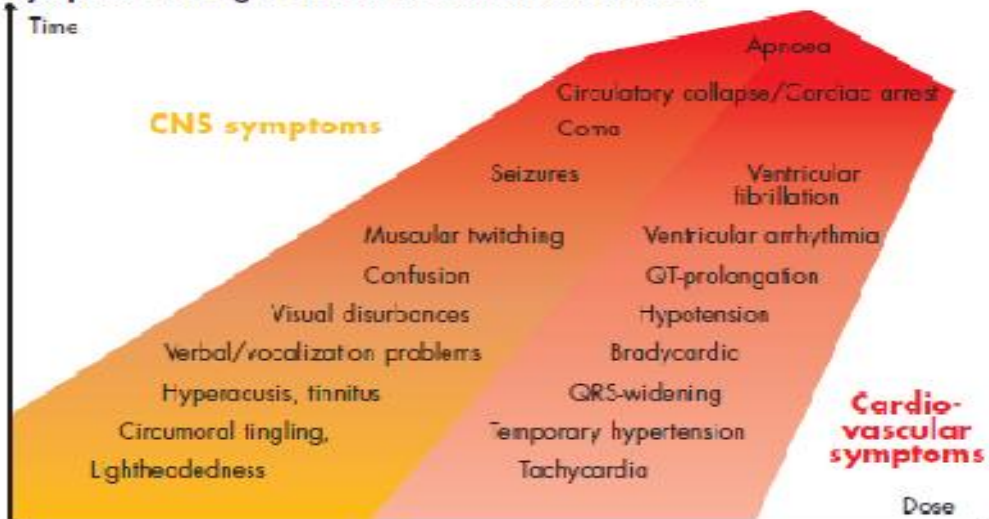
Pneumothorax, horner syndrome, phrenic nerve palsy, nerve injury (rare), accidental intravenous injection of the local anesthetics.

COMPLICATIONS OF PERIPHERAL NERVE BLOCK

- **Systemic toxicity of the local anaesthetic:** The most common reason being unintended intravascular injection. This risk can be minimised by
 - Adhering to the recommended dosages
 - Repeated aspiration and fractional injection
 - Slow injection, observing and maintaining verbal contact with the patient

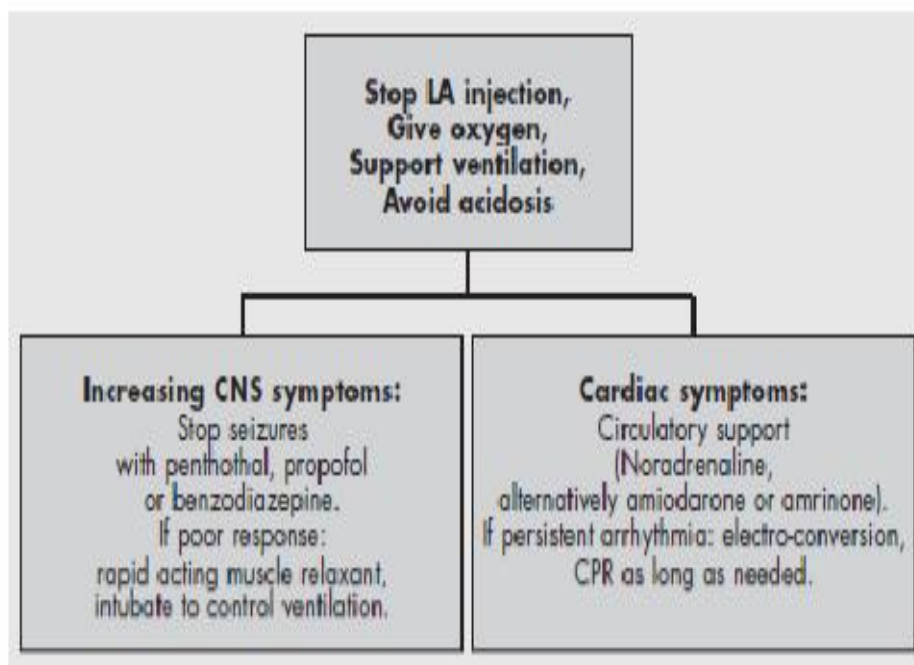
- **Nerve damage (extremely rare):** this risk can be minimised by
 - Trying to avoid paresthesia when inserting the needle.
 - Correct use of a suitable nerve stimulator ($\geq 0.3 - 0.5$ mA/ 0.1 ms).
 - The use of atraumatic needles.
- **Hematoma:** this can be prevented by avoiding blocks in the presence of a clinically manifest coagulation disorder or in a patient on treatment with anticoagulations.
- **Infection** (especially when using continuous techniques): The most sensitive indicator of infection is tenderness at the point of catheter entry. This can be prevented by aseptic needle insertion and regular planned checks of the catheter insertion site (at least once a day) and it requires immediate removal of catheter.

Symptoms and signs of local anaesthetic intoxication



A relative small dose of local anaesthetic, if accidentally injected intravascularly, may lead directly to seizures with both respiratory and cardiovascular problems, depending on drug and patient conditions.

Treatment of local anaesthetic intoxication



Treatment of Systemic Local Anesthetic Toxicity

- Get help and call for 20% lipid emulsion.
- Perform airway management. Hyperventilate with 100% oxygen.
- Abolish the seizures
- Perform cardiopulmonary resuscitation
- Epinephrine-controversial; may have to use higher doses than recommended in Advanced Cardiac Life Support (ACLS).
- Consider using vasopressin to support circulation
- Alert the nearest facility having cardiopulmonary bypass capability.
- Perform lipid emulsion treatment (for a 70-kg adult patient):
 - Bolus 1.5 mL/kg intravenously over 1 minute (about 100 mL)
 - Continuous infusion 0.25 mL/kg per minute (about 500 mL over 30 minutes)
 - Repeat bolus every 5 minutes for persistent cardiovascular collapse.
 - Double the infusion rate if blood pressure returns but remains low.
 - Continue infusion for a minimum of 30 minutes.

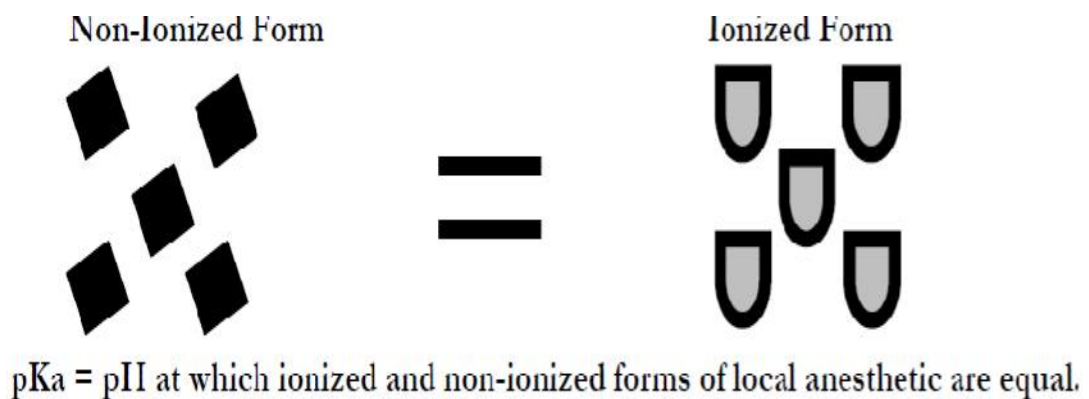
PHARMACOLOGY

LOCAL ANESTHETICS

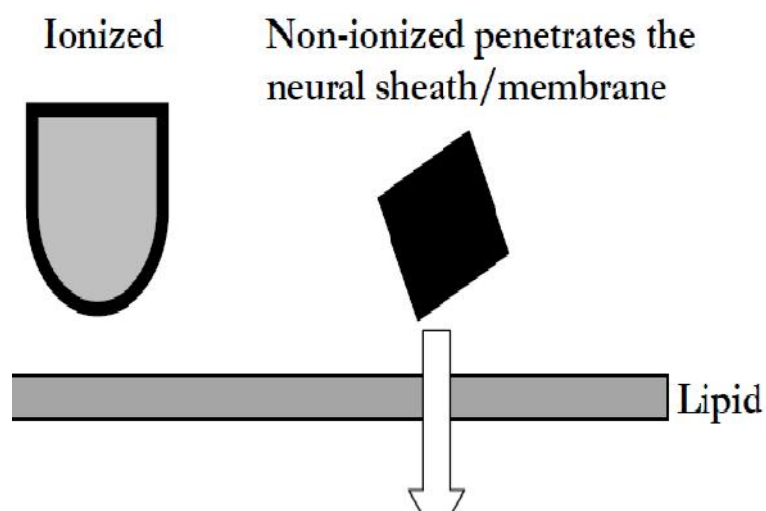
Local anesthetics are drugs used to prevent or relieve pain in specific regions of the body without loss of consciousness. Local anesthetics block pain sensation by blocking nerve conduction²³.

MECHANISM OF ACTION

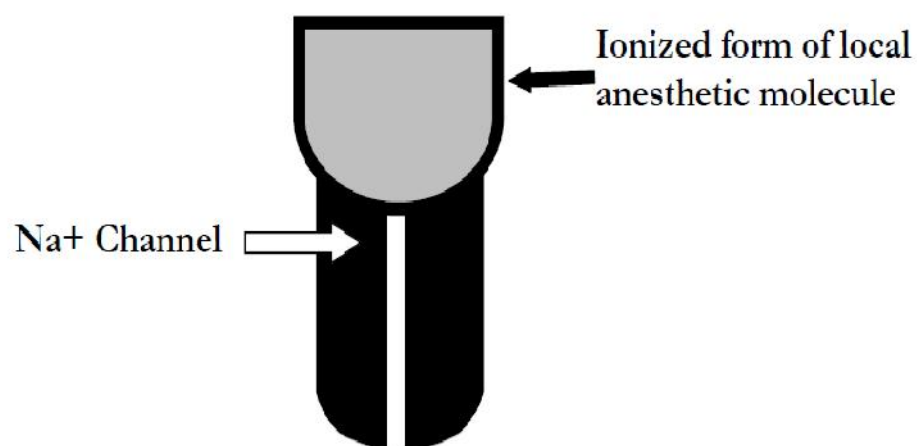
Local anesthetics exist as weak base, exist in a equilibrium between unionised and ionised form²³.



Nerve membranes are lipid membranes. Unionised form of local anesthetic enters the nerve membrane. Here the drug is ionised and it is the ionised form which blocks the nerve conduction, hence local anesthetic action is strongly dependent on local PH.(alkaline –good, acidic- bad).



Ionized- binds with the sodium channel

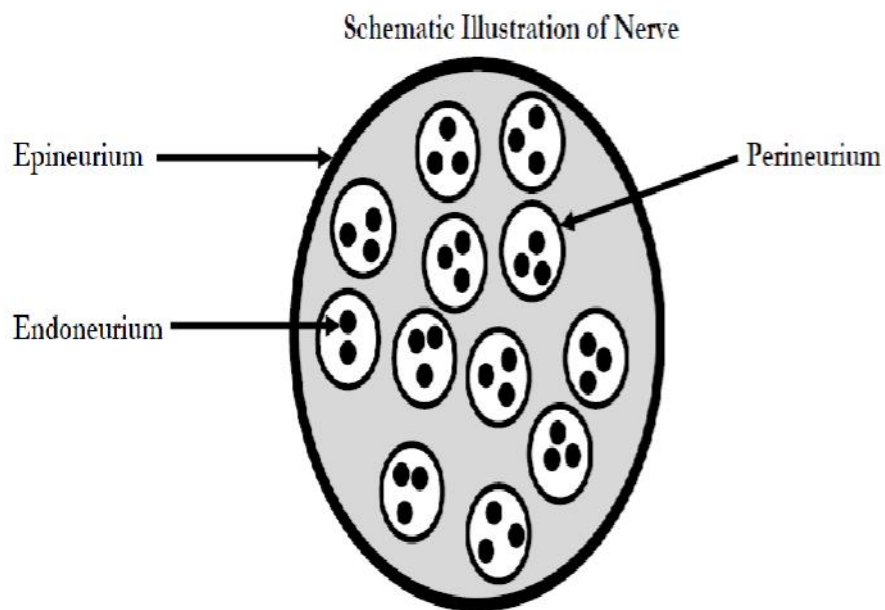


A peripheral nerve contains several axon bundles called fascicles.

Endoneurium is the connective tissue that covers an individual nerve.

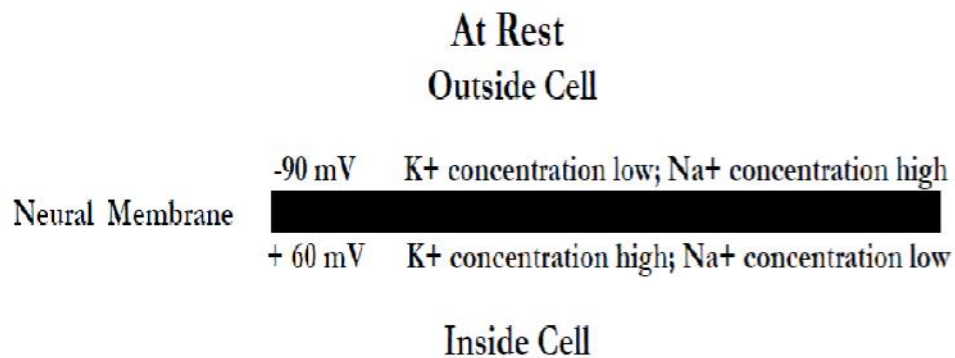
Perineurium is the connective tissue that covers each fascicle.

Epineurium is the connective tissue covering the entire nerve.

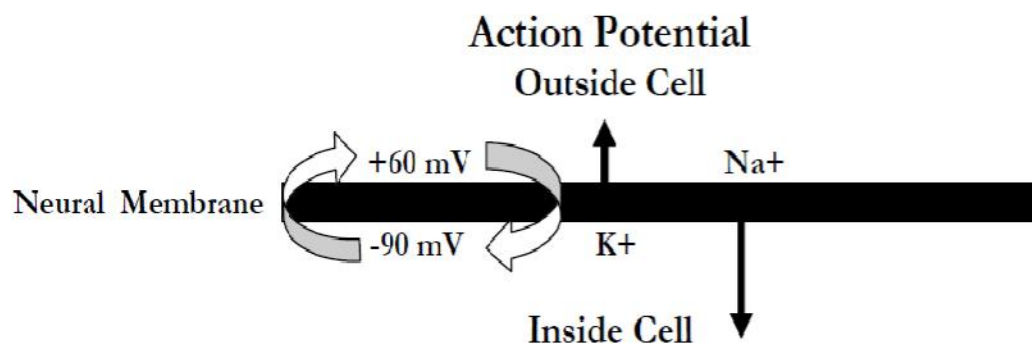


NERVE CONDUCTION PHYSIOLOGY

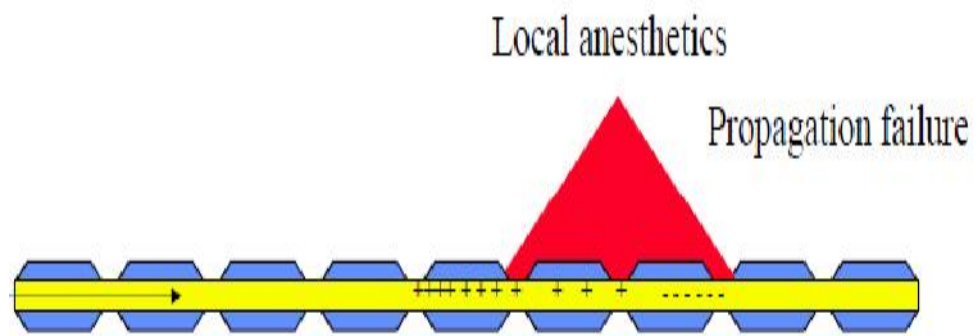
The neural membrane contains a voltage difference of +60 mV (inner) to -90 mV (outer). At rest the neural membrane is impermeable to Na^+ ions, and selectively permeable to K^+ ions. The Na^+/K^+ pump maintains the ion gradient.



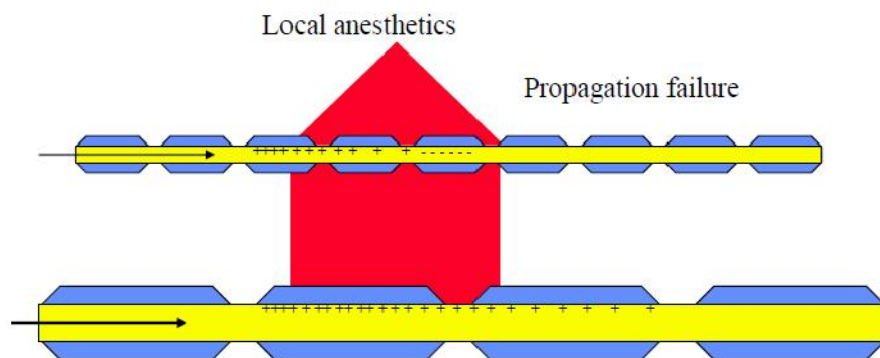
During an action potential, the nerve membrane switches its permeability from K⁺ to Na⁺, changing the membrane potential from -90 to +60 mV (negative to positive) and back again.



Local anesthetics reversibly bind to the voltage-gated Na^+ channel, block Na^+ influx, and thus block action potential and nerve conduction.



Small unmyelinated fibres, such as type C, that carry nociceptive signals (pain) are more susceptible to local anesthetic block than the larger fibres.



2. Local anesthetics reversibly blocks potassium channels and inhibit propagation.
3. Recent evidence has highlighted the diverse actions of local anesthetics. Not only do they block the sodium and potassium ion channels, but also interact with G- coupled receptor proteins, muscuranic receptor and endothelial nitric oxide. Attachment of local anesthetics to G- coupled receptor protein linked to lysophosphatilic acid , it attenuates neutrophils ,macrophage and monocyte function.

CLASSIFICATION OF LOCAL ANESTHETICS

AMIDES	ESTERS
BUPIVACAINE	BENZOCAINE
LIDOCAINE	COCAINE
ETIDOCAINE	PROCAINE
LEVOBUPIVACAINE	TETRACAINES
MEPIVACAINE	CHLORPROCAINE
PRILOCAINE	
ROPIVACAINE	

BUPIVACAINE

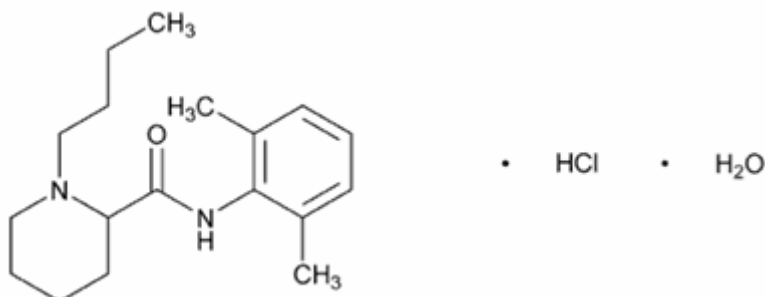
It is a long acting amino amide class of local anesthetic. It is a member of the series of n-alkyl substituted piperidyl xylidines first synthesised by Ekenstam in 1957, and was used clinically in 1963.

Chemical name : (2RS)-1-Butyl-N-(2,6-dimethylphenyl)piperidine-2-carboxamide hydrochloride monohydrate.

PHYSICAL PROPERTIES

It appears as a white, crystalline powder or colourless crystals. It is soluble in water, freely soluble in ethanol (96%), slightly soluble in chloroform and in ether. Bupivacaine has a pKa of 8.1 and is more lipid soluble than lignocaine.

CHEMICAL STRUCTURE



BUPIVACAINE

MECHANISM OF ACTION :

Bupivacaine reversibly inhibit the peripheral nerve conduction by blocking voltage gated sodium and potassium channels .

PHARMACOLOGICAL PROPERTIES

Molecular weight	PKa	Protein binding	Partition coefficient	Vd	T1/2	clearance	onset	duration
288	8.1	95	28	1.0	210	8.3	moderate	long

PHARMACOKINETICS

ABSORPTION: All Amides show a biphasic absorption pattern , with an initial rapid phase followed by a slow phase. Following the rapid entry of local anesthetics into the venous circulation, pulmonary extraction limits the concentrations of drug that reaches the systemic circulation for the distribution to the coronary and cerebral circulation. For bupivacaine this first pass pulmonary extraction is dose dependent, suggesting that the uptake process rapidly becomes saturated.

DISTRIBUTION : More widely distributed in the tissue .

METABOLISM : Liver

CLEARANCE: A small percentage of the bupivacaine is excreted unchanged in urine.

PLACENTAL TRANSFER: Protein binding determines the rate and diffusion of the local anesthetics across the placenta. Bupivacaine , which is highly protein bound (about 95%) , has an umbilical vein / maternal arterial ratio of 0.3%.Acidosis in fetus , as occurs during prolonged labour , can result in accumulation of local anesthetics in the fetus by ion trapping²⁴.

PHARMACODYNAMICS

Systemic absorption of local anesthetics produces effects on the cardiovascular and central nervous systems.

CARDIOVASCULAR SYSTEM

At blood concentrations achieved with normal therapeutic doses, changes in cardiac conduction, excitability, refractoriness, contractility, and peripheral vascular resistance are minimal. However, toxic blood concentrations depress cardiac conduction and excitability, which may lead to atrioventricular block, Ventricular arrhythmias, and cardiac arrest,

sometimes resulting in fatalities. In addition, myocardial contractility is depressed and peripheral vasodilation occurs, leading to decreased cardiac output and arterial blood pressure. Recent clinical reports and animal research suggest that these cardiovascular changes are more likely to occur after unintended intravascular injection of Bupivacaine. Therefore, incremental dosing is necessary²⁴.

CENTRAL NERVOUS SYSTEM

Following systemic absorption, local anesthetics can produce central nervous system stimulation, depression, or both. Apparent central nervous stimulation is manifested as restlessness, tremors progressing to convulsions, followed by depression and coma progressing ultimately to respiratory arrest. However, the local anesthetics have a primary depressant effect on the medulla and on higher centers. The depressed stage may occur without a prior excitation²⁴.

INDICATIONS AND USES

Local or regional anesthesia or analgesia for surgery, oral surgery procedures, diagnostic and therapeutic procedures, and for obstetric procedures.

Routes of administration	Concentrations
Local infiltration	0.25%
Peripheral nerve block	0.25% and 0.5%
Retro bulbar block	0.75%
Sympathetic block	0.25%
Lumbar epidural	0.25%,0.5%, and 0.75%(0.75% not for obstetric patients)
Caudal	0.25% and 0.5%

Bupivacaine is manufactured in a concentration of 0.25% or 0.5% for peripheral nerve block. It has a long duration of action, from 2 to 5 hours following a single epidural injection and up to 12 hours after peripheral nerve blocks. The onset of the blockade is slower than with lignocaine, especially when anaesthetising large nerves.

When used in low concentrations (2.5 mg/mL or less) there is less effect on motor nerve fibres and the duration of action is shorter. Low concentrations may, however, be used with advantage for prolonged pain relief, e.g. in labour or postoperatively. The plasma concentration of bupivacaine depends upon the dose, the route of administration and the vascularity of the injection site. The addition of a vasoconstrictor such as adrenaline may decrease the rate of absorption and prolong the duration of action.

Following injection of bupivacaine solutions for caudal, epidural or peripheral nerve block, peak plasma levels of bupivacaine are reached within 30 to 45 minutes and decline to insignificant concentrations during the next 3 to 6 hours.

Intercostal blocks give the highest peak plasma concentration due to rapid absorption (maximum plasma concentrations in the order of 1 to 4 mg/L after a 400 mg dose), while subcutaneous abdominal injections give the lowest plasma concentrations.

Epidural and major plexus blocks are intermediate. In children rapid absorption (plasma concentrations are in the order of 1 to 1.5 mg/L after a dose of 3 mg/kg) is seen with caudal block. Absorption may be slowed by the addition of adrenaline.

CONTRADICTIONS

1. Paracervical block.
2. Allergy to amides group of drugs.

ADJUVANTS

Adjuvants are used in peripheral nerve block to

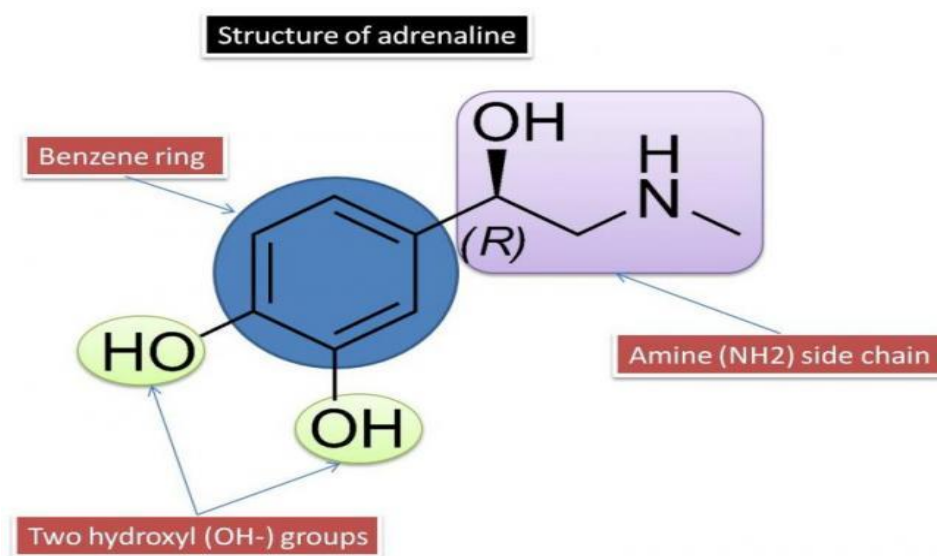
1. Increase the duration of action.
2. Increase the onset of action.
3. Increase the quality and density of block.
4. Improve the overall analgesic effect.

WHAT HAS BEEN TRIED?

- Opioids (fentanyl⁴, sufentanil, morphine, buprenorphine⁵, nalbuphine, tramadol³)
- Epinephrine²
- Sodium bicarbonate
- Steroids (dexamethasone, methylprednisolone)
- α -agonists (clonidine^{2,6}, dexmedetomidine⁷)
- Midazolam
- Neostigmine
- Verapamil
- Magnesium
- Ketamine
- NSAIDs (ketorolac) and Lysine acetylsalicylic acid.

EPINEPHRINE

Vasoconstrictors are among the most common additives to local anesthetic solutions. The duration of a local anesthetic is proportional to the duration of time that the local anesthetic remains in contact with a nerve; therefore, a vasoconstrictor can prolong the duration by decreasing vascular resorption. Epinephrine is the most commonly used vasoconstrictor in combination with local anesthetics²⁴.



Epinephrine (adrenaline), in concentration of 1 in 200,000 or 1 in 400,000 is sometimes added to local anesthetics to reduce vascular adsorption and potential local anesthetic toxicity²⁴.

Preparation of 1 in 200,000 epinephrine (adrenaline) involves dilution of 0.1 ml of 1 in 1,000 (0.1 mg) epinephrine or 1ml of 1 in 10,000 (0.1 mg) to 20 ml volume of the local anesthetics ; 1 in 400,000 dilutions are made with half of the above doses of adrenaline²⁴.

Epinephrine containing solutions contain a reducing agents, sodium metabisulphite , to prevent oxidation of the epinephrine .In addition , small amount of preservative and fungicide may be added.

The vasoactive effect of epinephrine (adrenaline) is greater with the more lipid soluble amides .

DRUG	DOSE	WITH EPINEPHRINE
LIGNOCAINE	3 mg/kg	7mg/kg
BUPIVACAINE	2.5mg/kg	3mg/kg

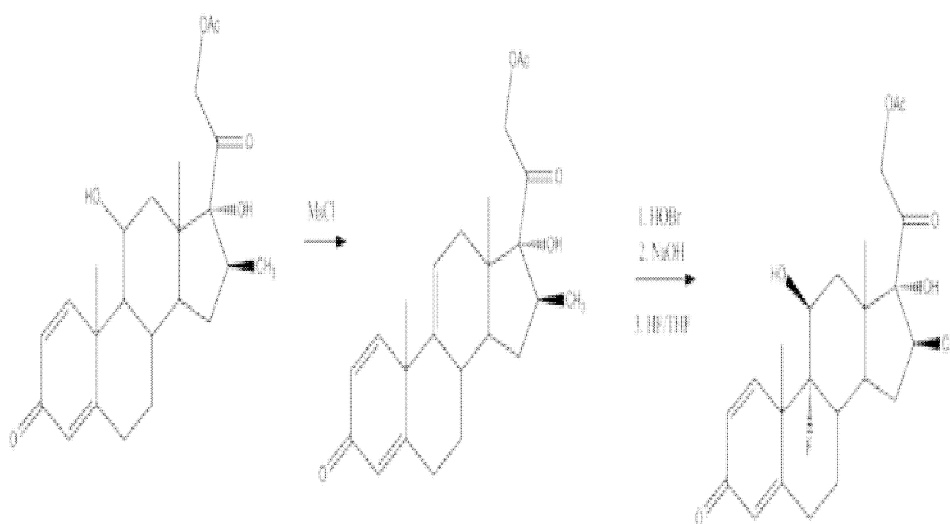
Epinephrine, by local vasoconstriction at the site of block minimises the systemic absorption of bupivacaine. This reduces the risk of bupivacaine toxicity.

DEXAMETHASONE

Dexamethasone is a longer acting , potent and highly selective glucocorticoid. It is 25 times more potent than cortisol in its glucocorticoid activity.

SYNTHESIS AND CHEMICAL STRUCTURE

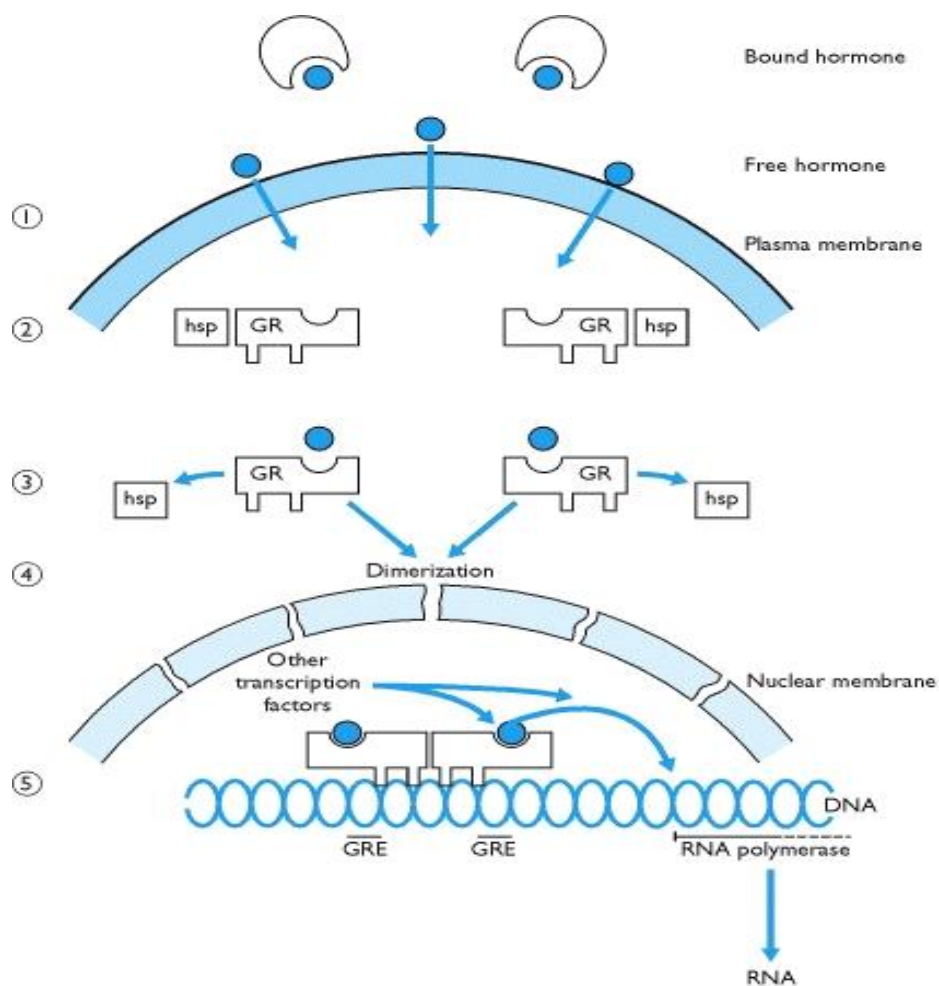
The 16 β - methyl prednisolone acetate is dehydrated to form a 9,11-dehydro derivative. This then reacts with a source of hypobromite such as basic N-bromosuccinimide, to form the 9 α -bromo-11 β -hydrin derivative which is then ring-closed to an epoxide. A ring-opening reaction along with hydrogen fluoride in tetrahydrofuran gives dexamethasone.



Dexamethasone

MECHANISM OF ACTION

Steroids penetrate the cells and bind to a high affinity cytoplasmic receptor protein. A structure change occurs in the steroid receptor complex that allows its migration into nucleus and binding to specific sites on the chromatin. This causes transcription of specific m-RNA resulting of protein synthesis. This process takes at least 30-60 min . Effects of steroids are not immediate , and once appropriate proteins are synthesized , effects persist much longer than the steroid itself²⁵.



The glucocorticoid receptor is very widely distributed. Some actions of glucocorticoid are exerted more rapidly but mediated by a different mechanism not involving the protein synthesis.

MAINTENANCE OF HOMEOSTASIS

Permissive and protective effects of glucocorticoids are critical for the maintenance of homeostasis during periods of stress. These actions of glucocorticoids are complementary and permit the individual to effect an appropriate stress response to stimuli and to maintain the homeostasis.

PERMISSIVE ACTIONS

Permissive actions occur at a low physiological steroid concentration and serve to prepare the individual for responding to stress. These actions of glucocorticoids maintain the basal activity of the hypothalamic pituitary axis by providing negative feedback and setting a threshold for a response to stress²⁵.

PROTECTIVE ACTIONS

Protective actions occur when high concentrations of steroids exert the anti-inflammatory and immune-suppressive effects. This response prevents the host defence mechanisms that are activated during stress. Other important protective actions of glucocorticoids include redirection of metabolism to meet energy needs during stress²⁵.

PHARMACODYNAMICS

1. Carbohydrate metabolism

Long term steroid use promotes glycogen deposition in the liver by inducing hepatic glycogen synthetase and promoting gluconeogenesis.

They inhibit glucose utilisation by the peripheral tissue. This along with increased glucose release from the liver results in hyperglycemia, resistance to insulin, and a diabetes like state²⁶.

2. Protein metabolism

On chronic use they cause protein breakdown and amino acid mobilisation from peripheral tissues which is responsible for side effects like muscle wasting, lympholysis and loss of osteoid from bone and thinning of skin. The amino acids which are mobilised are funnelled into liver and are used up in gluconeogenesis; as a result excess urea is produced leading to negative nitrogen balance. Glucocorticoids are thus catabolic²⁶.

3. Fat metabolism

Long term use of glucocorticoids has a permissive action. They promote lipolysis due to glucagon, growth hormone, adrenaline and thyroxine. Further, cyclic AMP induced breakdown of triglycerides is

enhanced. Fat depots in different areas respond differently and redistribution of body fat occurs. Subcutaneous tissue over the extremities loses fat which is deposited over the face, neck and shoulder causing moon face, fish mouth, and buffalo hump²⁶.

4. Calcium metabolism

Long term steroids inhibit intestinal absorption and enhance renal excretion of calcium. There is also loss of calcium from bone indirectly due to loss of osteoid leading to negative calcium balance .Spongy bones like vertebrae, ribs are more sensitive²⁶.

5. Water excretion

Effect on water excretion is independent of action on sodium transport. In adrenal insufficiency, the capacity to excrete a water load is markedly reduced –such patients are prone to water intoxication from intravenous infusions. Glucocorticoids also enhance secretory activity of renal tubules²⁶.

6. Cardiovascular system

Glucocorticoids restrict capillary permeability; maintain tone of arterioles and myocardial contractility. They have a permissive effect on the pressor action of adrenaline and angiotensin²⁶.

They also play a permissive role in the development of hypertension; when considered for long term use , they should be used cautiously in hypertensive patients.

7. Central nervous system:

Mild euphoria is common with pharmacological doses of glucocorticoids. This is a direct effect on brain , independent of relief of disease symptoms ; sometimes progresses to cause increased motor activity , anxiety , insomnia or depression²⁶.

Glucocorticoids also maintain the level of sensory perception and normal level of excitability of neurones. In high dose, glucocorticoids lower the seizure threshold, hence caution is required when prescribed for epileptics.

8. Skeletal muscles

Optimal level of corticosteroid is needed for normal muscular activity . Weakness occurs in both hypo and hypercorticism, but the causes are different. In hypocorticism, diminished work capacity and weakness are primarily due to hypodynamic circulation. In hypercorticism, excess mineralocorticoid action causing hypokalemia and weakness²⁶.

9. Gastrointestinal system

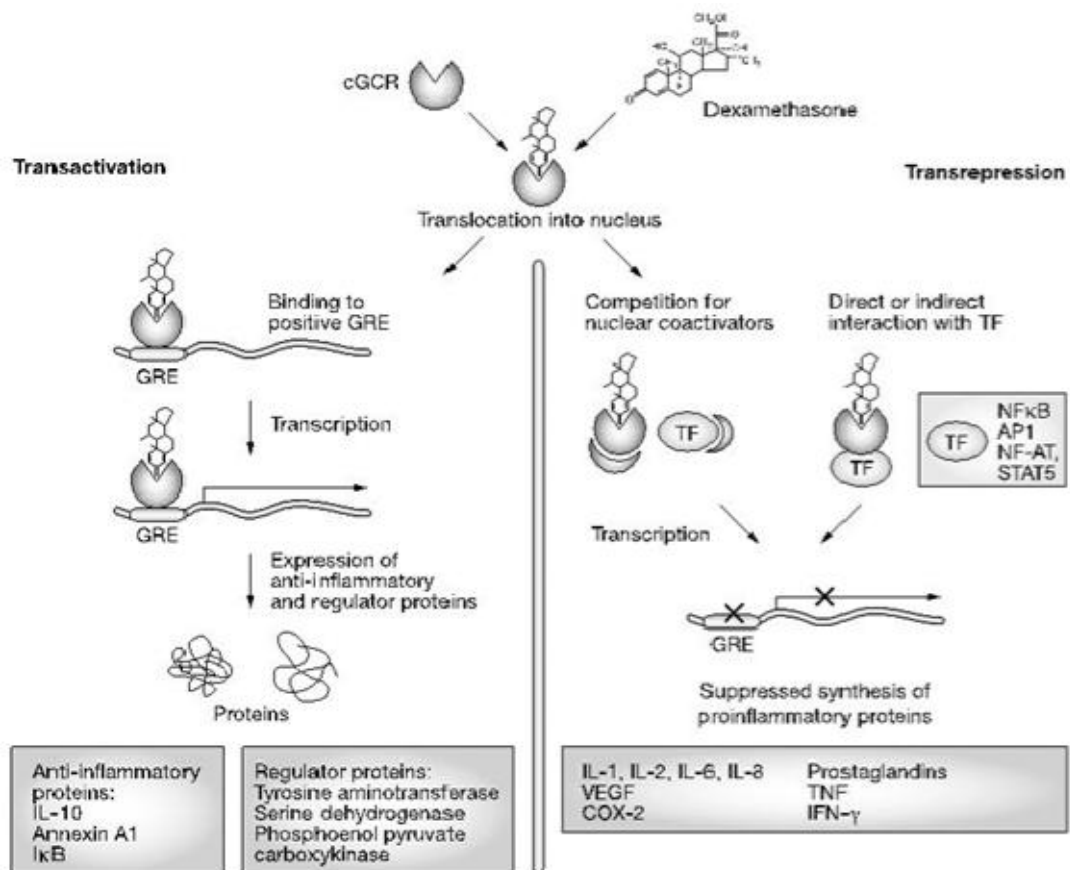
Secretion of gastric acid and pepsin is increased, peptic ulcer may be aggravated²⁶.

10. Lymphoid tissue and blood cells

Glucocorticoids enhance the rate of destruction of lymphoid cells ; but in man the effect on normal lymphoid tissue is modest. Glucocorticoids increase the number of RBCs, decrease lymphocytes , eosinophils and basophils .This is not due to destruction of these cells but due to their sequestration in tissues. Blood counts come back to normal after 24 hrs²⁶.

11. Anti Inflammatory responses

Mechanisms include decreased production of interleukin-1, interleukin-2, interleukin-3, interleukin-6, tumor necrosis factor, granulocyte monocyte colony forming factor and gamma interferon. This results in suppression of fibroblast proliferation and T-lymphocyte chemotaxis is interfered with . The other effects include reduction of increased capillary permeability, local exudation, cellular infiltration, phagocytic activity and late responses like capillary proliferation, collagen, and fibroblastic activity and ultimately scar formation. The action is direct and local therefore topical use is possible²⁶.



The most important mechanism appears to be the limitation of recruitment of inflammatory cells at the local site. Glucocorticoids are only palliative, they do not remove the cause of inflammation.

12. Immunological and allergic response

Glucocorticoids impair immunological competence. They suppress all types of hypersensitisation and allergic phenomenon . The clinical effect appears to be due to suppression of recruitment of leukocytes at the site of contact with antigen and of inflammatory response to immunological injury.

They cause greater suppression of Cell mediated immunity in which T cells are primarily involved, eg. Delayed hypersensitivity and graft rejection . This is the basis of use in autoimmune diseases and organ transplantation. Factors involved may be inhibition of Interleukin-1 release from macrophages , inhibition of Interleukin-2 formation and action .T cells proliferation is not stimulated . Corticosteroids also cause suppression of natural killer cells²⁶ .

PHARMOKINETICS

Bioavailability	80-90%
Protein binding	77%
Metabolism	hepatic
Half-life	190 min
Excretion	Urine(65%)

USES

1. POST OPERATIVE NAUSEA AND VOMITING (PONV)

Dexamethasone along with ondansetron prevents postoperative nausea and vomiting only when administered near the beginning of surgery, probably by reducing the surgery induced inflammation due to inhibition of prostaglandin synthesis as for as 24 hours²⁸.

2. DAY CARE SURGERY

Steroids decrease post operative nausea and vomiting , postoperative pain , by decreasing prostaglandins and increasing the level of endorphins²⁸.

3. ANAPHYLACTIC AND ALLERGIC REACTIONS

Steroids because of their delayed onset are not the drug of choice in treating anaphylactic reactions . They are usually used as an adjuvant to adrenaline. Manifestations of allergic diseases that are of limited duration like hayfever , serum sickness, urticaria, contact dermatitis, drug reactions, bee stings, and angioneurotic edema can be suppressed by adequate doses of corticosteroids. Topical steroids having the potent anti – inflammatory effects are the main stay of allergic therapy^{28,29,30}.

These medications interfere with the inflammatory response and multiple aspects of allergic cascade. Corticosteroids work by inhibiting the production of inflammatory cytokines and chemokines, thus decreasing inflammation and cellular edema, and cellular recruitment to site of disease.

4. SEPTIC SHOCK AND SEVERE SEPSIS

Patient with sepsis have an unrecognised adrenal insufficiency with an incidence of as high as 28%. Steroid therapy is associated with success in the withdrawal of vasopressors²⁸.

5. POSTOPERATIVE ANALGESIA

Glucocorticoids inhibit the phospholipase enzyme that is necessary for the inflammatory chain reaction along both the cyclooxygenase and lipoxygenase pathways. As a result, glucocorticoids may be effective in decreasing postoperative pain. It has been shown that dexamethasone given intravenously before induction of anaesthesia for anorectal surgery was associated with shortened time to discharge from ambulatory surgery²⁸.

Routes of administration of steroids include parenteral, nerve blocks and central neuraxial blockade. Mode of analgesia is ill defined

but thought to be due to their anti-inflammatory action which decreases the inflammatory mediators responsible for pain sensation. They also increase endorphins and thus cause mood elevation.

6.PERIOPERATIVE REPLACEMENT THERAPY

One of the important component of stress response in our body is increase in the circulating levels of cortisol in response to surgical stimulus .This response is essential to avoid metabolic , electrolyte , fluid balance and hemodynamic instability. It is necessary in patients with hypo adrenocortism or in patients with present or previous history of steroid intake. The dose and duration remains controversial. Patients taking steroids for transplant procedures should receive the same dose of steroids with no extra dose of steroids required during the perioperative period. There is no fixed protocol for steroid replacement therapy^{28,29}.

7. ANTI INFLAMMATORY AND HYPER REACTIVE AIRWAY

Steroids have anti-inflammatory actions at higher doses. Perioperative uses include hyper-reactive airways due to asthma, foreign body, and trauma , anaphylactic reactions like drug allergies, blood transfusion reactions and transplantation of solid organs^{28,29}.

8.CEREBRAL EDEMA

Corticosteroids in larger doses are of value in the reduction or prevention of cerebral edema and the resulting increases in intracranial pressure that may accompany tumors and metastatic lesions in the brain .Dexamethasone, with its minimal mineralocorticoid activity, is frequently used to decrease cerebral edema and associated increase in intracranial pressure^{28,29}.

9.POST INTUBATION LARYNGEAL EDEMA

Dexamethasone, 0.1-0.2 mg/kg intravenously can be administered for the treatment of post intubation laryngeal edema .But the efficacy of steroids for this condition has not been confirmed²⁸.

10.RESPIRATORY DISTRESS SYNDROME

Administration of steroids at least 24 hours before delivery decreases the incidences and severity of respiratory distress syndrome in neonates born between 24 and 34 weeks gestation. Dexamethasone improves the pulmonary and neurodevelopment outcome of a low birth weight infant at risk of bronchopulmonary dysplasia.²⁸

11.OTHERS

To test adrenal pituitary axis function, congenital adrenal hyperplasia, aspiration pneumonitis, lumbar disc disease, immunosuppression, arthritis, collagen vascular disease, cutaneous disorders, ocular inflammation²⁸.

DEXAMETHASONE AS ANALGESIC

Why dexamethasone would prolong regional anaesthesia is of much discussion?

The mechanism of action of corticosteroids producing analgesia is still controversial. Their effects are suspected to be mediated by anti-inflammatory and immunosuppressive effects.

There are some authors who believe that the nerve block prolonging effect of dexamethasone is due to its local action. Steroids produce analgesia by blocking the transmission in nociceptive C-fibres and thus suppressing the ectopic neuronal discharge. For example, local application of methylprednisolone blocks the transmission of C fibres but not the alpha and Beta fibres.

This effect is reversible, probably suggesting a direct membrane effect. Steroids may bring this effect by altering the function of the potassium channels in the excitable neurons. The analgesic effect of dexamethasone may be related to its local action. It should be emphasized that blockade is not produced by corticosteroid alone for peripheral nerve blocks, but these steroids may potentiate the effect of local anesthetic drugs in the modulation of the potassium channels in the excitable neuron.

MATERIALS AND METHODS

The study was carried out in the Department of Anesthesiology, ESIC MC AND PGIMSR, K. K Nagar, Chennai from March 2013 to AUGUST 2014.

STUDY DESIGN: - Prospective randomized control trial

PARTICIPANTS

Participants were recruited from the patients posted for upper limb surgeries in ESIC MC AND PGIMSR .The protocol was approved by the Ethical committee of ESIC MC AND PGIMSR. After departmental approval and obtaining written informed consent from the patients, American Society of Anaesthesiologist (ASA) physical status I or II patients of either sex, aged 18-60 years scheduled for elective or emergency surgeries of the upper limb under supraclavicular brachial plexus block were included in this study.

INCLUSION CRITERIA

All patients undergoing upper limb surgeries (arm, forearm, hand surgeries) under supraclavicular block between 18 to 60 years of age and belonging to ASA physical status I or II were included in this study.

EXCLUSION CRITERIA

- Any bleeding disorder and patient on anticoagulants
- Contralateral lung injury and rib fracture
- Pre-existing neuropathy involving the surgical limb
- Local infection at the injection site
- History of allergy to local anaesthetic
- Patients with a history of peptic ulcer disease, hepatic or renal failure (contraindication to steroids)
- Pregnant women
- Systemic use of corticosteroids for 2 weeks or longer within 6 months of surgery

The patients were explained about the study in detail and written informed consent was taken.

BASELINE EVALUATION

The initial evaluation consisted of detailed history and general physical and systemic examination. Data recorded include information on age, weight, height, body mass index (BMI) and blood pressure measurement.

LABARATORY METHODS

Baseline Laboratory investigations included Complete hemogram , Blood urea nitrogen , Blood sugar , liver function tests , chest X-ray and Electrocardiogram.

INTERVENTION

RANDOMIZATION : The patients were then randomly assigned to either the group D (study group) and the group B (control group) as determined by the slips in box method.

All patients included in the study on arrival to the operating room had an 18 G intravenous (IV) line started on the contralateral arm and standard monitors like pulse oximetry, non invasive blood pressure (NIBP) and electrocardiogram(ECG) were attached. Baseline pulse rate , blood pressure , oxygen saturation were recorded.

LOCAL ANESTHETIC PREPARATION

Local anesthetics drugs are taken with the use of 20 ml syringes and kept in sterile bowl.

GROUP D

DRUGS	TOTAL VOLUME -28 ml
BUPIVACAINE 0.5% with 1.25ml of 1 in 10000 epinephrine	25 ml
DEXAMETHASONE 8mg	2 ml
DISTILLED WATER	1 ml

GROUP B

DRUG	TOTAL VOLUME – 28 ml
BUPIVACAINE 0.5% with 1.25ml of 1 in 10000 epinephrine	25 ml
DISTILLED WATER	3 ml

Oxygen is administered at the rate of 4L/min through face mask. Midazolam 1mg iv given. A set containing nerve stimulator, insulated 50 mm needle, ECG electrode, Two 20 ml syringes, Skin marker pencil, one tuberculin syringe of 1 ml, one stainless sterile bowl for local anesthetic drugs preparation, and sterile gauze pieces. Supraclavicular block by a classical approach was given using a nerve stimulator.

TECHNIQUE

Supraclavicular block –Classical approach (nerve-stimulator technique)

1. Patient was placed in a dorsal recumbent position without a pillow with arms at his / her sides and head turned to side opposite to the one being blocked. A small pad was placed below bilateral shoulders.



The patient was then asked to lower the shoulders and flex the elbow, so that the forearm rests on his/her lap. The wrist was supinated so the palm of the hand faces the patient's face.

2. Part of the neck was aseptically cleaned and draped.
3. The operator stood on the side to be blocked so for a left side block the palpation was done with the left hand and the needle was manipulated with the right and vice versa.
4. The lateral (posterior) border of the sternocleidomastoid (SCM) muscle was identified and followed distally to the point where it met the clavicle. The point of needle entrance was about 1 in (2.5 cm) lateral to the insertion of the SCM in the clavicle or one "thumb breadth" lateral to the SCM. Palpation of the subclavian artery at this site confirms the landmark. The palpating index finger was placed at this site.

5. Local infiltration with 1ml of 2% lignocaine was given.



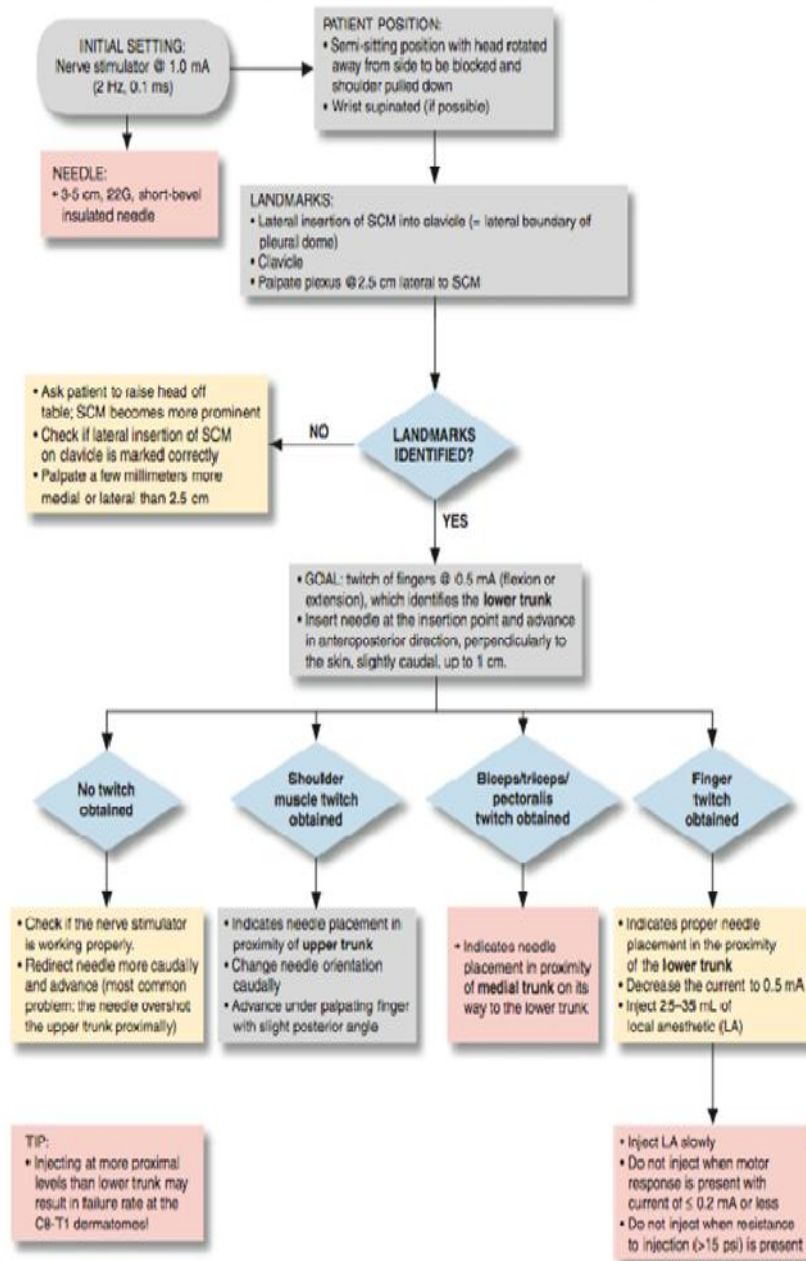
6. An insulated needle of length 50 mm was used to perform this technique. The needle was connected to nerve locator by the electrodes and was properly grounded with the help of ECG lead. The stimulation is started with an intensity of 2.0 mA and a pulse width of 100 μ s. Once the desired response is obtained i.e a muscle twitch of the fingers that is clearly visible, the current was gradually decreased upto 0.6mA. If the response is obtained at 0.4mA also, then the needle was repositioned again so as to get response at 0.6mA but not at 0.4mA.

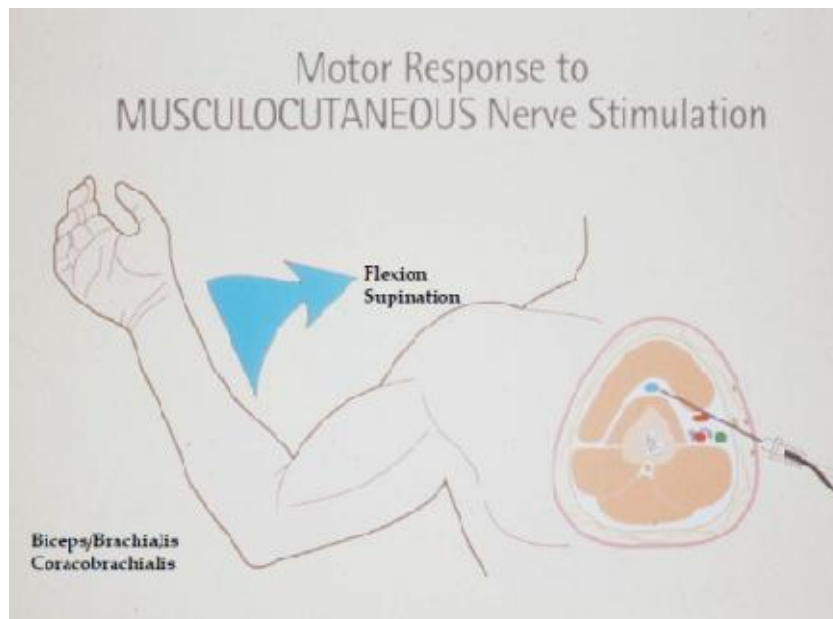
7. If we did not get an adequate response or if repositioning of the needle is necessary, the needle was withdrawn and the penetration angle is adjusted in the antero-posterior plane.

As a goal we aimed to elicit an isolated muscle twitch in all fingers either in flexion or extension.

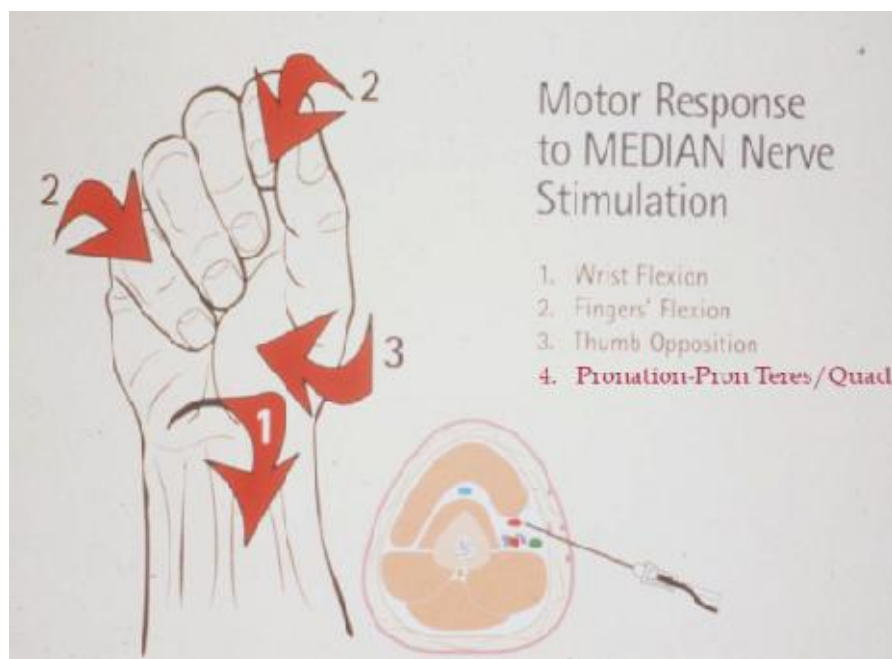
Wrist flexion and extension of the fingers was taken as acceptable responses and the current gradually reduced to between 0.2 to 0.5 mA . The total volume of the anesthetic solution was injected at an incremental dose of 5ml each, preceded by negative aspiration. 3-min massage was performed to facilitate an even drug distribution.

NERVE STIMULATOR-GUIDED SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK: DECISION-MAKING ALGORITHM

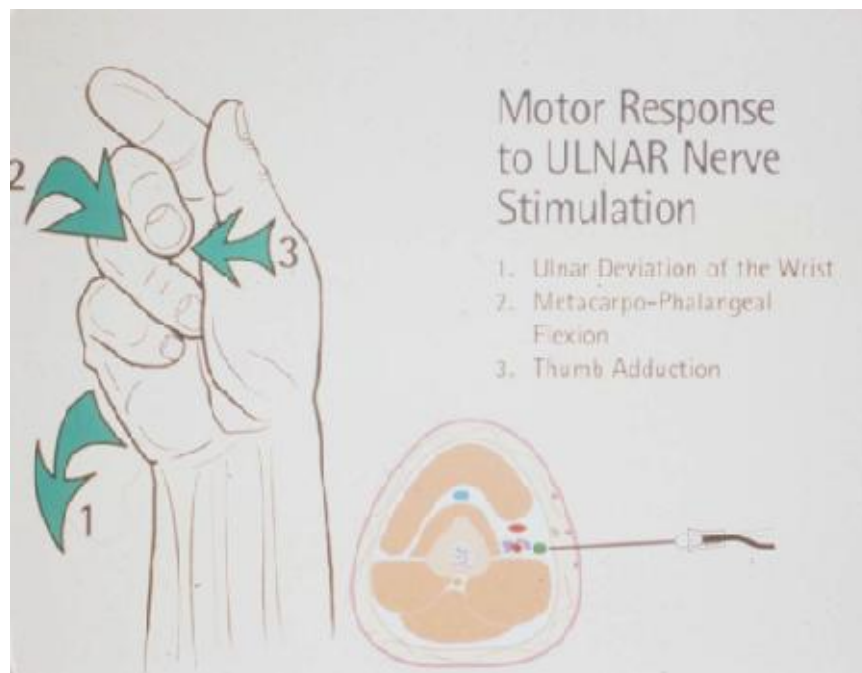




FLEXION AT ELBOW (Musculocutaneous nerve)

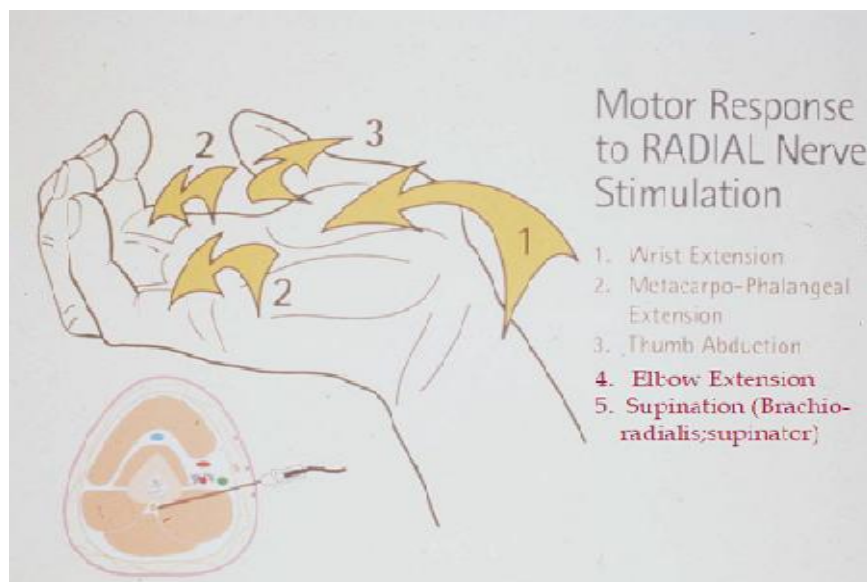


WRIST FLEXION AND FINGER FLEXION (median nerve)



ULNAR DEVIATION OF WRIST AND THUMB

ADDUCTION(ulnar nerve)



WRIST EXTENSION AND THUMB

ABDUCTION(radial nerve)

Vital parameters were observed throughout the procedure every 15 minutes as usual and recorded.

BLOCK EVALUATION

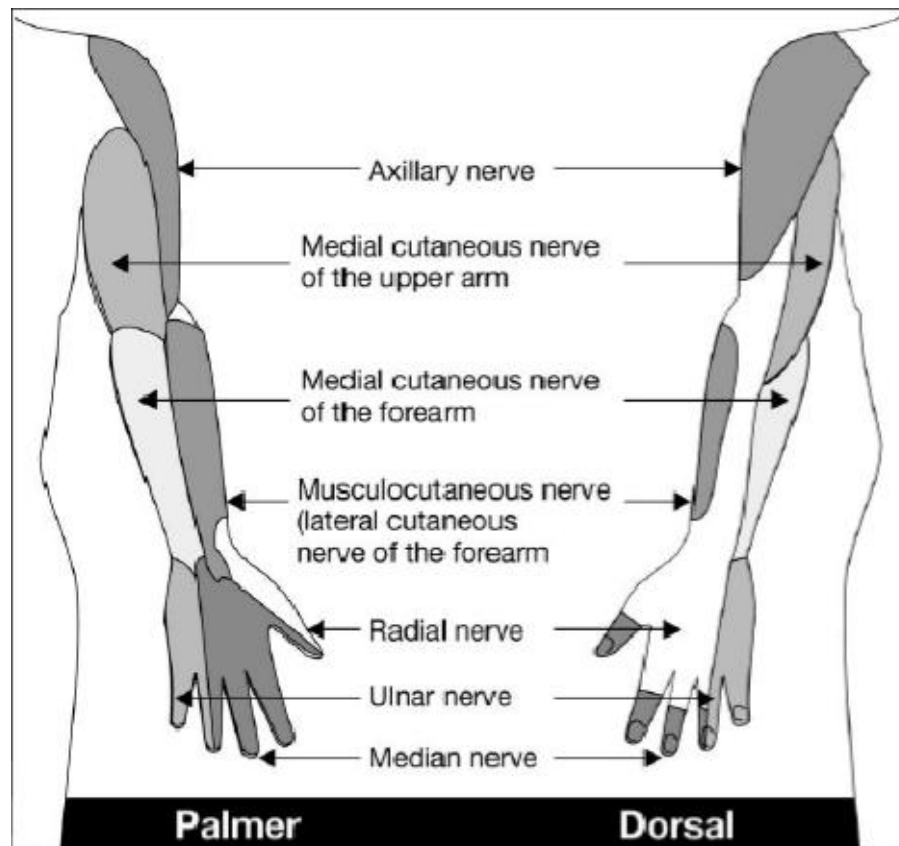
Sensory block was assessed by pinprick method using a end of a 27 gauge needle at 0, 2, 4, 6, 8 and every 2 minutes till 30 minutes in the median ulnar, radial , musculocutaneous nerve distribution.

GRADES OF SENSORY BLOCKADE¹⁷:

Grade 0: Sharp pin felt.

Grade 1: Analgesia, dull sensation felt

Grade 2: Anaesthesia, no sensation felt



The onset time of sensory blockade was defined as the time interval between the end of local anesthetic injection and loss of sensation to pin prick (grade 1).

MOTOR BLOCK¹⁷

Motor block was measured at 0, 5, 10, 15, 20, 25, and 30 minutes by assessing

1. extension of elbow and wrist (radial nerve)
2. opposition of the thumb and index finger (median nerve)

3.opposition of the thumb and small finger(ulnar nerve)

4.flexion at the elbow (musculocutaneous nerve)

MOTOR BLOCK- MODIFIED BROMAGE SCALE.

Grade 0: Normal motor function with full flexion and extension of elbow, wrist and fingers.

Grade 1: Decreased motor strength with ability to move the fingers only.

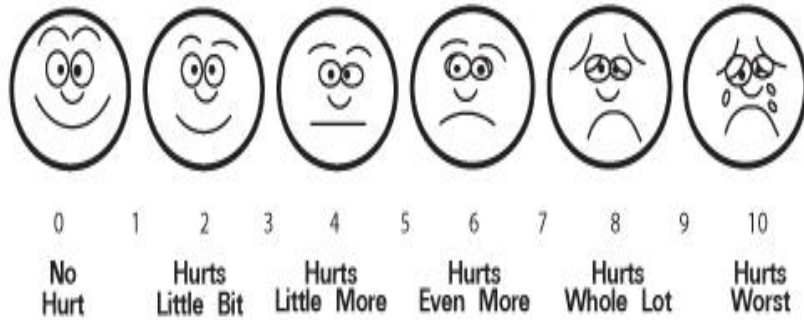
Grade 2: Complete motor block with inability to move the fingers.

The onset time of motor blockade was defined as the time interval between the end of local anesthetic injection and paresis (grade 1) in the distributions of all peripheral nerves respectively.

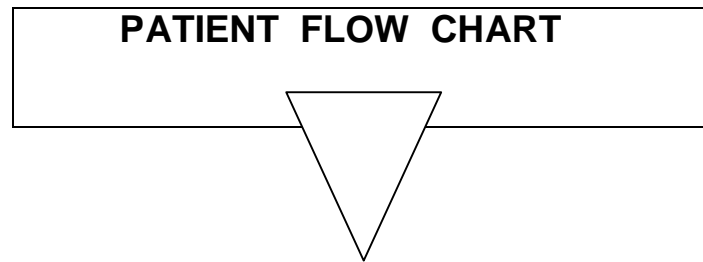
The surgery was allowed to proceed when complete anesthesia is achieved. The duration of surgery in each case was noted.

The duration of analgesia was defined as the time between onset of analgesia to first pain perception by patient. The duration of analgesia was noted according to the visual analogue score (VAS) for pain for every hour for first 10 hours and then every second hourly till 24 hours.

Wong Baker Face Scale

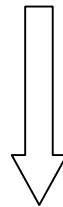


- When the patients began to experience the worst pain (VAS ≥ 4), it is considered that analgesic action of the drugs is terminated and rescue analgesic (Intramuscular Diclofenac 1 mg/kg) given. The duration of analgesia was noted for every patient.



ASSESSMENT CLINIC ASA I AND ASA II (18-60 YEARS)

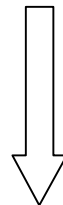
INFORMED CONSENT



***VISUAL ANALOGUE
SCORE EXPLAINED***

ON THE DAY OF SURGERY 80 PATIENTS(upper limb surgery)

RANDOMISATION



SLIPS IN BOX

METHOD

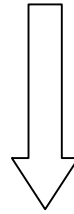
GROUP D

GROUP B

***Patient shifted inside the operation theatre by the trained
personel with a trolley .***

INSIDE THE THEATRE

WHO CHECK LIST



MONITORS CONNECTED

Intravenous line started with 18 G needle

Airway cart and rescue measures kept

Group D

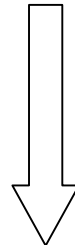
***Bupivacaine 0.5% with 1.25ml
of 1 in 10000 Epinephrine – 25ml***

***Dexamethasone – 8mg
Distilled Water- 1ml***

Group B

***Bupivacaine 0.5% with 1.25ml
of 1 in 10000 Epinephrine – 25ml***

Distilled water -3 ml



SUPRACLAVICULAR BLOCK – CLASSICAL METHOD

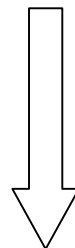
(Nerve stimulator)

Onset of motor block

(modified Bromage scale)

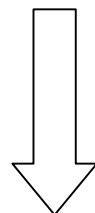
onset of sensory block

(pin prick method)



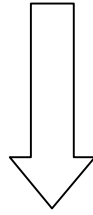
Vital parameters monitored every 15 minutes

Duration of surgery noted



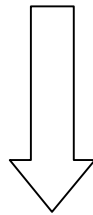
POSTOPERATIVE PERIOD

Patient followed up with VAS



VAS > 4

Rescue analgesia



Diclofenac injection i.m

DURATION OF ANALGESIA NOTED

OBSERVATION AND RESULTS

Discrete categorical data were presented as n (%); continuous data were given as mean \pm SD. For normally distributed data, t-test was applied for comparison between two groups. For experimental data (onset of sensory and motor blockade, duration of analgesia and visual analogue score), independent t-test was applied for comparison between two groups. For multiple comparisons (between different time intervals) One Way ANOVA test was applied. All statistical tests were two-sided and performed at a significance level of $\alpha=0.05$. Analyses were conducted using SPSS for Windows (version 15.0; SPSS Inc., Chicago, IL, USA).

A total of eighty patients who were given supraclavicular block for various upper limb surgeries completed this randomized controlled study. Of these 80 patients, 40 were in group D (study group) and 40 were in group B (control group).

TABLE1
COMPARISON OF BASELINE DATA BETWEEN
THE TWO GROUPS

VARIABLE	GROUP B n=40	Group D n=40	p value
Age in years (Mean±S.D)	34.6 ± 11.44	37.45 ± 11.71	0.27
Weight in kg (Mean±S.D)	65.15 ± 5.47	65.9 ± 5.11	0.53
Height in cms (Mean±S.D)	166 ± 5.65	166.52 ± 8.06	0.71
BMI in kg/m² (Mean±S.D)	23.6 ± 1.31	23.78 ± 1.24	0.63
Sex distribution			
a. Males	35	33	0.54
b. Females	05	07	
ASA			
a. I	26	30	0.34
b. II	14	10	
Level of surgery			
a. Elbow and above elbow	10	11	0.51
b. Forearm	25	19	
c. Hand	05	10	
Type of surgery			
a. Elective	37	32	0.11
b. Emergency	03	08	

BMI-body mass index;ASA-American society of anaesthesiologist.

FIGURE 1
COMPARISON OF AGE BETWEEN THE TWO GROUPS

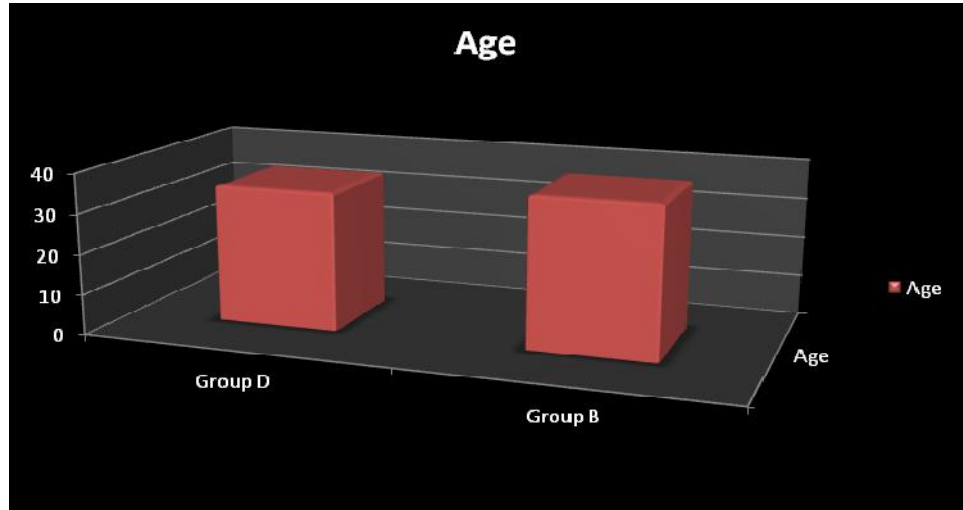


FIGURE 2
COMPARISON OF WEIGHT BETWEEN THE TWO GROUPS

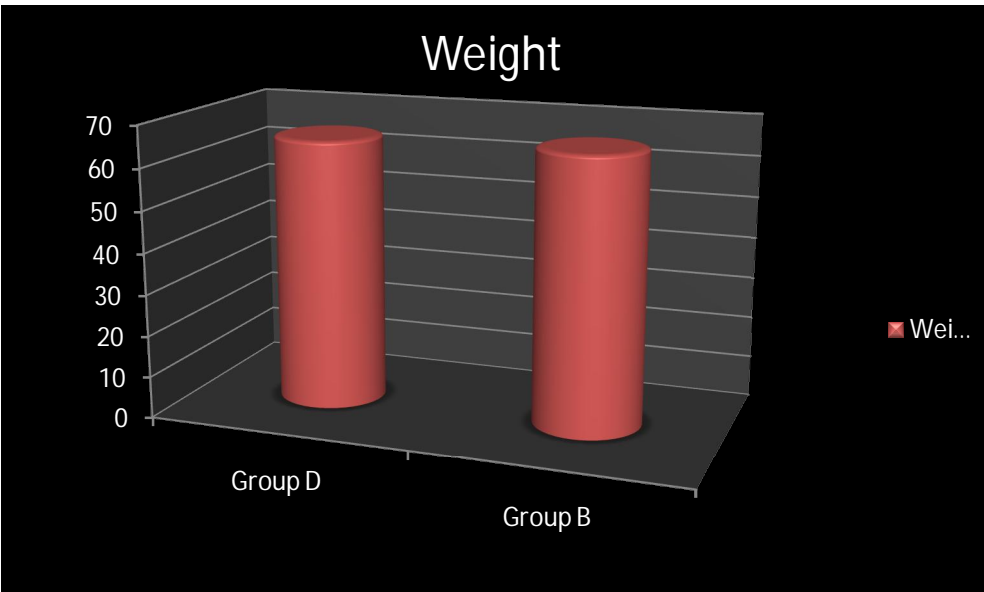


FIGURE 3
COMPARISON OF HEIGHT BETWEEN THE
TWO GROUPS

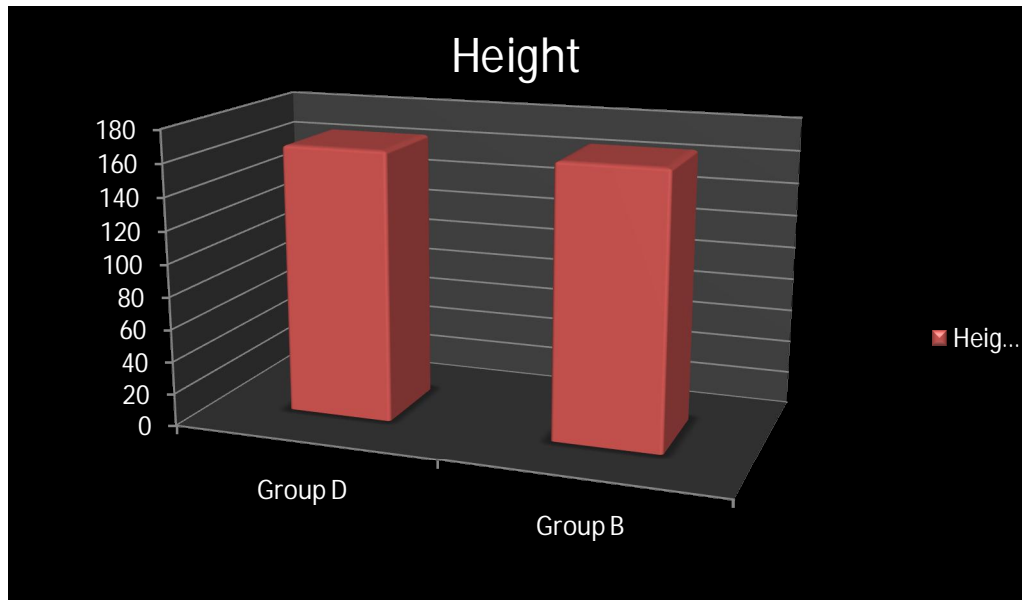


FIGURE 4
COMPARISON OF BMI BETWEEN
THE TWO GROUPS

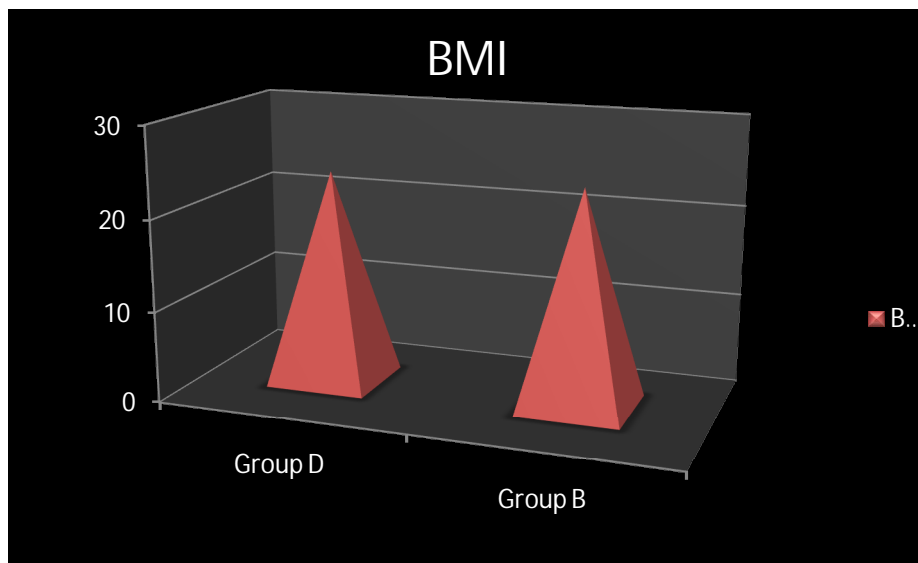


FIGURE 5
SEX DISTRIBUTION IN THE TWO GROUPS

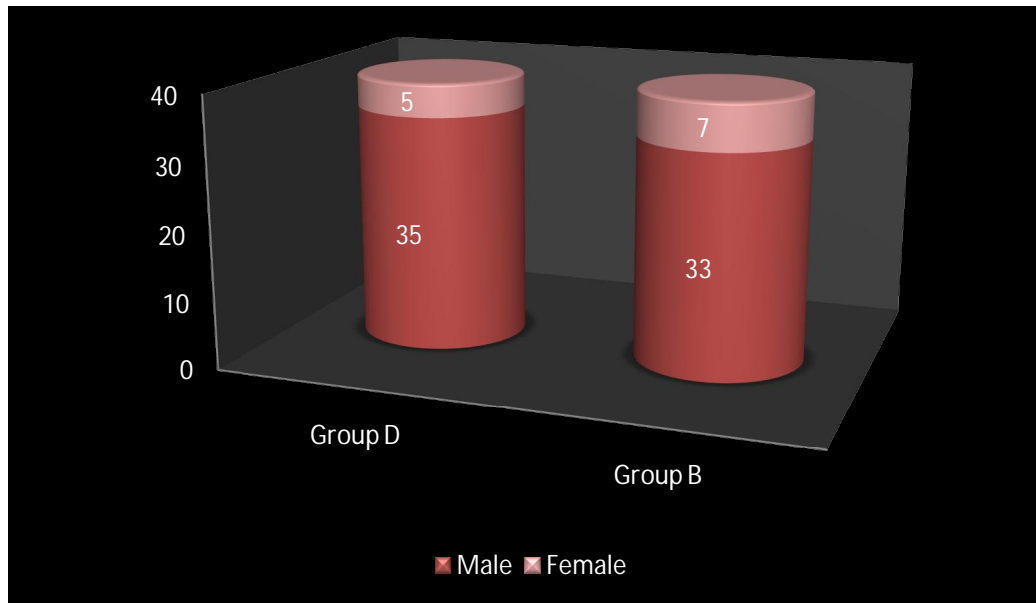
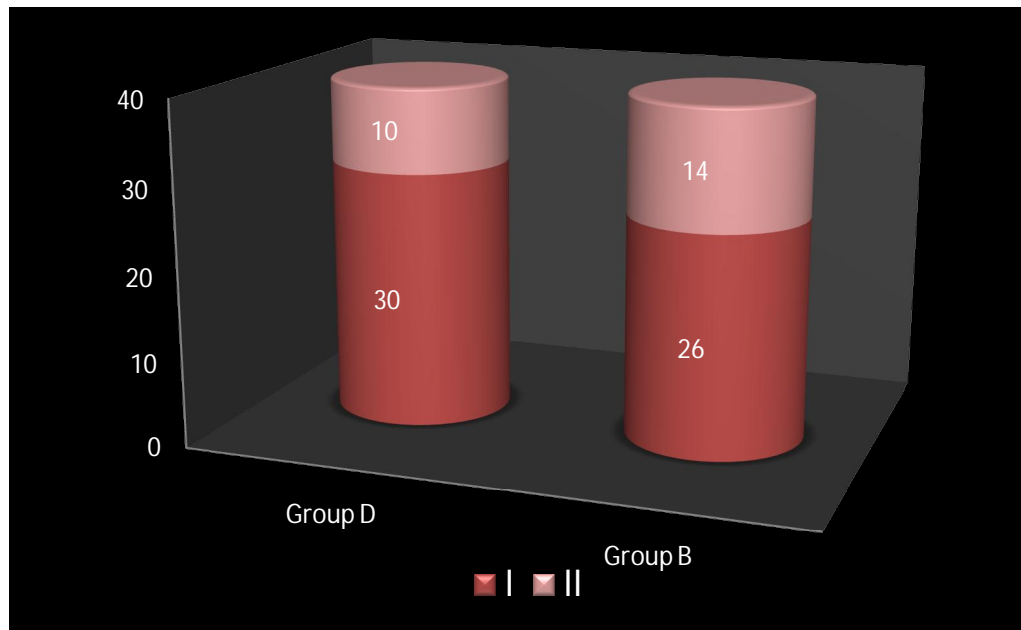


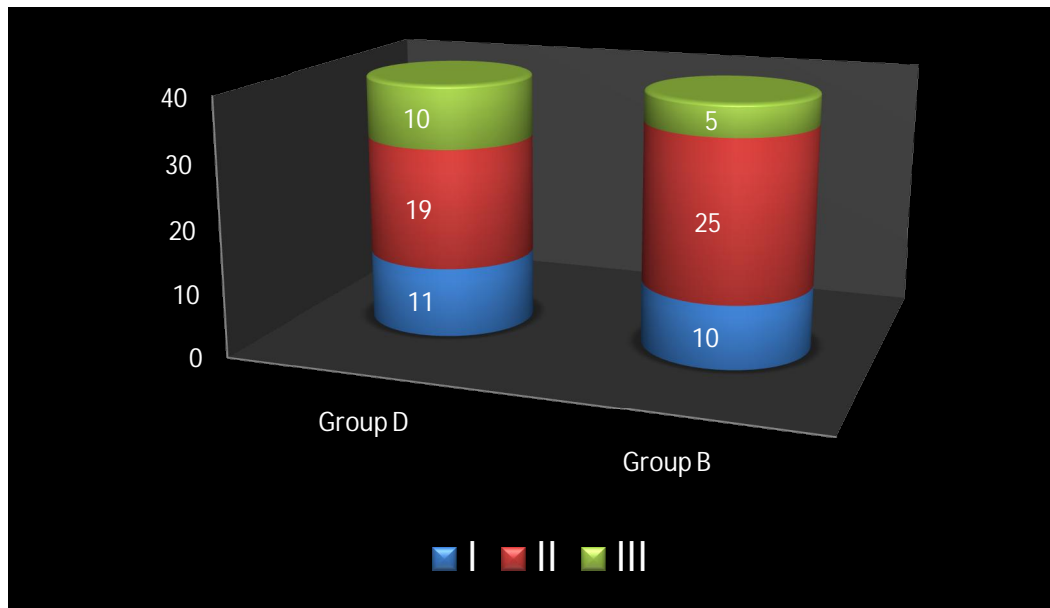
FIGURE 6
COMPARISON OF ASA CLASS BETWEEN
THE TWO GROUPS



In figure 6: I -ASA I, II- ASA II

FIGURE 7

COMPARISON OF LEVEL OF SURGERY IN 2 GROUPS



In figure 7:

I - Elbow and above elbow surgery

II - Fore arm surgery

III – Hand surgery

FIGURE 8
COMPARISON OF TYPE OF SURGERY BETWEEN
THE TWO GROUPS

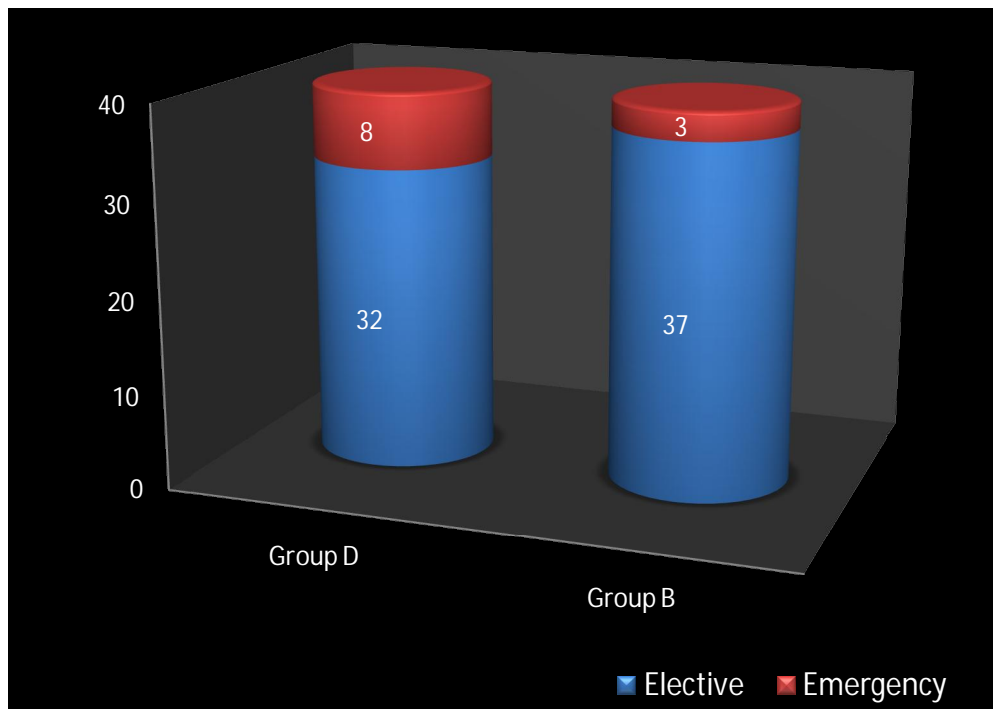


Table 1 and Figure 1- 8, clearly demonstrates that all the baseline data were comparable between the two groups of patients.($p>0.05$).

TABLE 2

**COMPARISON OF ONSET OF MOTOR AND SENSORY
BLOCKADE**

	GROUP B	GROUP D	p VALUE
	(Mean±S.D)	(Mean±S.D)	
ONSET OF MOTOR BLOCKADE (MIN)	11.5±3.43	7.5±3.2	< 0.0001*
ONSET OF SENSORY BLOCKADE(MIN)	13.6±6	4.2±0.99	< 0.0001*

*highly significant (p<0.0001)

FIGURE 9

**COMPARISON OF ONSET OF MOTOR BLOCKADE BETWEEN
THE TWO GROUPS**

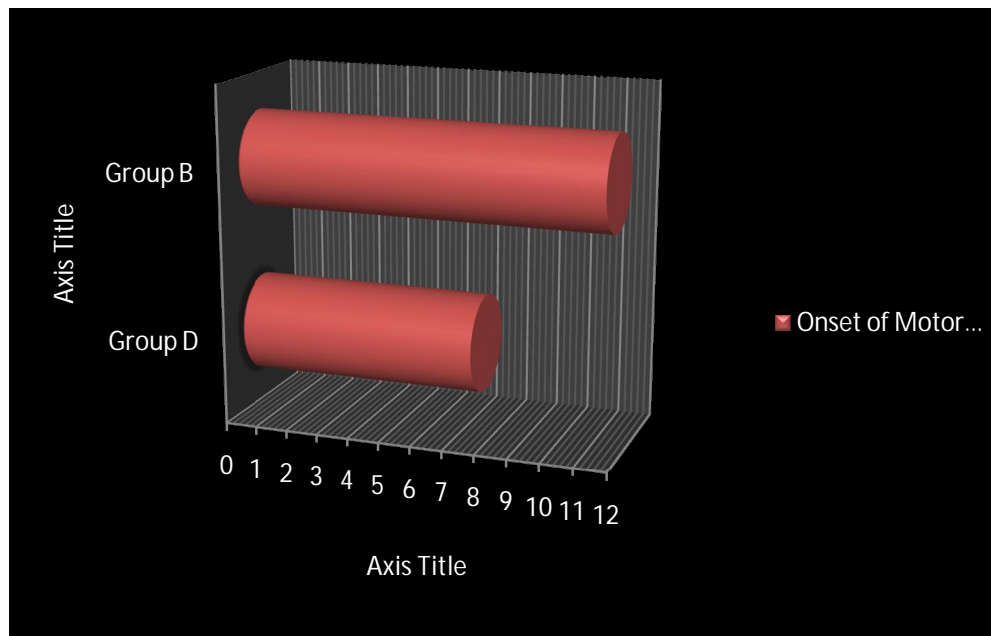


FIGURE 10

**COMPARISON OF ONSET OF SENSORY BLOCKADE OF
THE TWO GROUPS.**

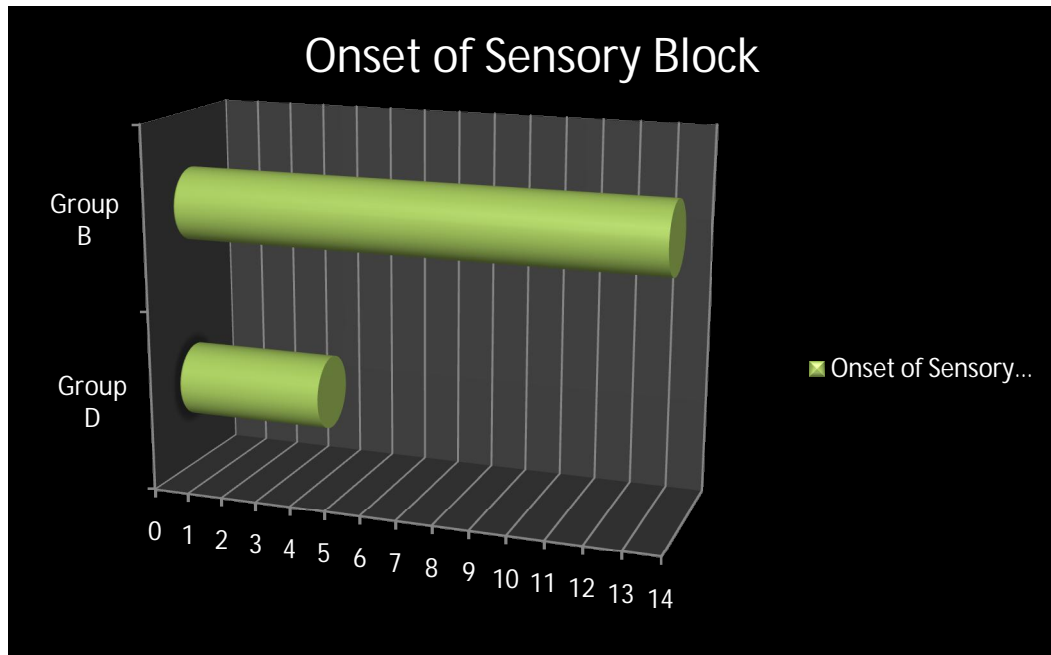


Table 2 and figure 9 and 10 show the comparison between the two groups with respect to onset of sensory and motor blockade. There was a significantly earlier onset ($p < 0.0001$) of both sensory and motor blockade in the group D (study group) as compared to group B (control group) .

TABLE 3

**COMPARISON OF PULSE RATE OF THE TWO GROUPS AT
DIFFERENT TIME INTERVALS OF THE STUDY**

Groups	Pre op	0 min	15 min	30 min	45 min	60 min	p value
Group D (beats/min)	87.85 ± 5.36	86.58 ± 5.82	87.4 ± 8.73	85.2 ± 8.2	85.18 ± 7.93	86.7 ± 8.6	0.511
Group B (beats/min)	91.9 ± 11.49	95.18 ± 7.71	94.25 ± 7.12	94.5 ± 7.36	93.15 ± 6.9	91.5 ± 7.52	0.257

FIGURE 11

**COMPARISON OF PULSE RATE OF THE TWO GROUPS AT
DIFFERENT TIME INTERVALS OF THE STUDY**

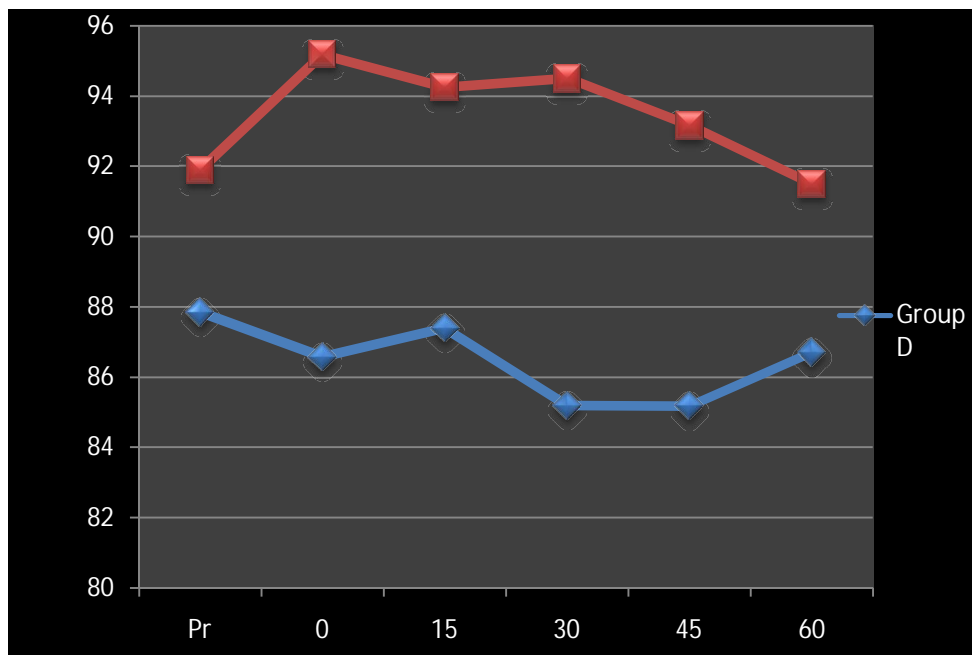


TABLE 4

**COMPARISON OF SYSTOLIC BLOOD PRESSURE
OF THE TWO GROUPS AT DIFFERENT TIME
INTERVALS OF THE STUDY.**

Groups	Pre op	0 min	15 min	30 min	45 min	60 min	P value
Group D (mm/Hg)	119.43 ± 13.22	119.15 ± 10.97	119.83 ± 9.75	118.03 ± 10.14	121.43 ± 10.34	118.1±10. 04	0.746
Group B (mm/Hg)	118.4 ± 13.21	121.83 ± 10.89	122.4 ± 10.79	121.05 ± 9.54	119.15 ± 9.32	116.22±9. 09	0.083

FIGURE 12

**COMPARISON OF SYSTOLIC BLOOD PRESSURE THE TWO
GROUPS AT DIFFERENT TIME INTERVALS OF THE STUDY**

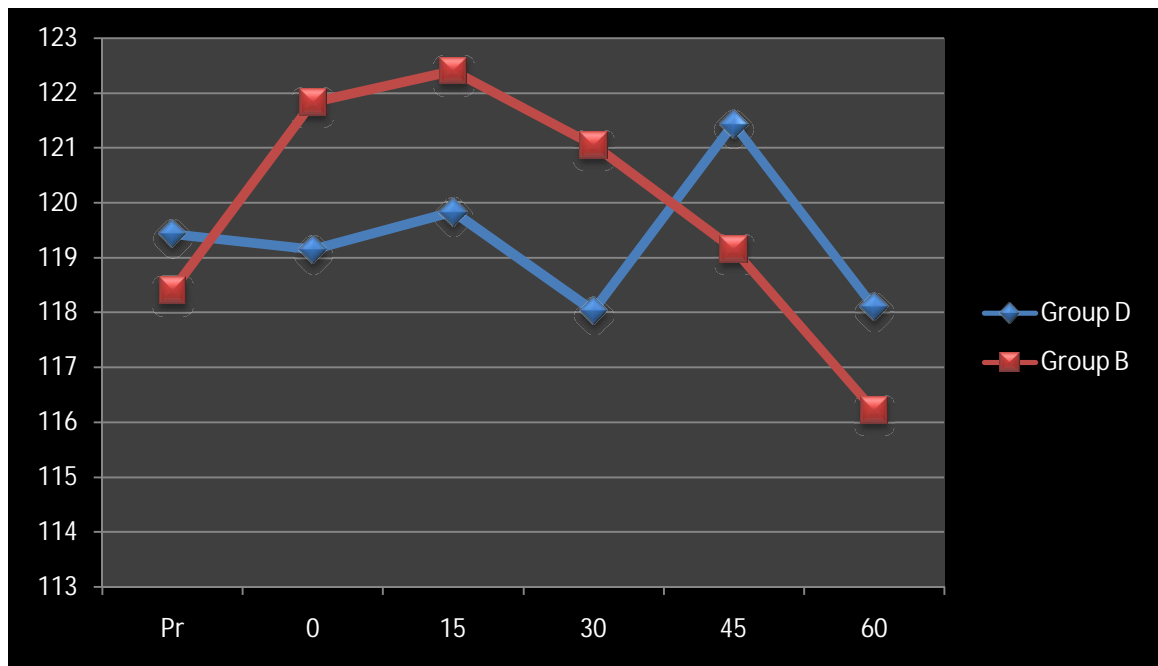


TABLE 5

COMPARISON OF DIASTOLIC BLOOD PRESSURE OF THE TWO GROUPS AT DIFFERENT TIME INTERVALS OF THE STUDY

Groups	Pre op	0 min	15 min	30 min	45 min	60 min	p value
Group D (mm/Hg)	74.13 ± 7.13	72.8 ± 4.63	72.53 ± 4.56	72.45 ± 3.57	73 ± 3.54	71.65 ± 3.92	0.305
Group B (mm/Hg)	73.85 ± 7.36	75.18 ± 6.88	73.03 ± 4.05	72.93 ± 3.88	72.38 ± 4.84	72.8 ±4.61	0.230

FIGURE 13

COMPARISON OF DIASTOLIC BLOOD PRESSURE OF THE TWO GROUPS AT DIFFERENT TIME INTERVALS OF THE STUDY

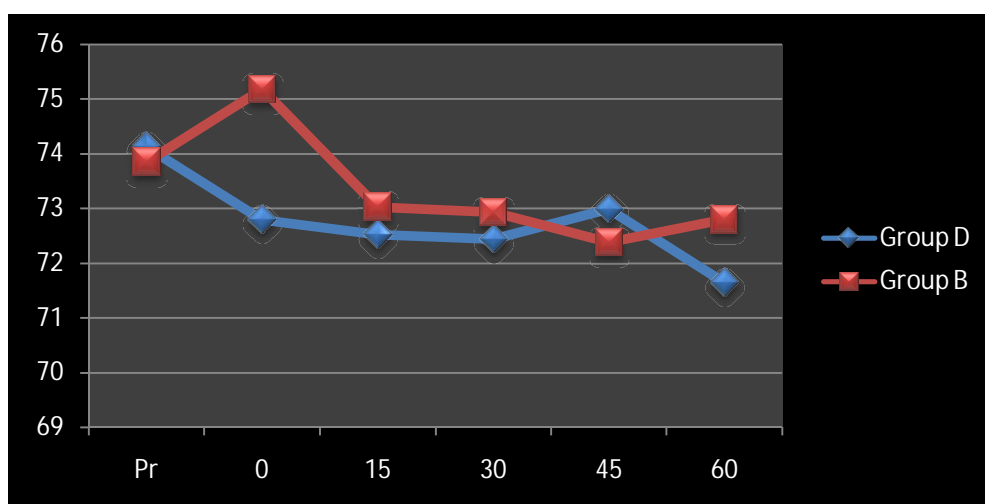


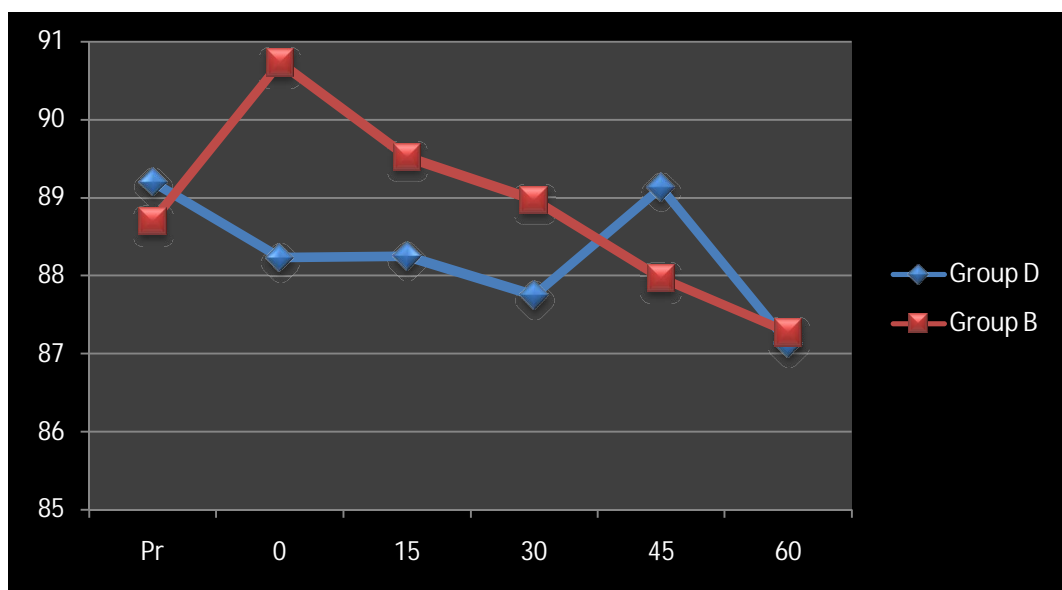
TABLE 6

**COMPARISON OF MEAN ARTERIAL PRESSURE (MAP) OF
THE TWO GROUPS AT DIFFERENT TIME INTERVALS OF
THE STUDY**

Groups	Pre op	0 min	15 min	30 min	45 min	60 min	p value
Group B (mm/Hg)	89.2 ± 8.08	88.23 ± 5,27	88.25 ± 4.71	87.75 ± 4.49	89.13 ± 4.67	87.13 ± 4.82	0.452
Group D (mm/Hg)	88.7 ± 8.15	90.73 ± 6.83	89.52 ± 5.04	88.97 ± 3.86	87.97 ± 5.78	87.27 ± 3.64	0.126

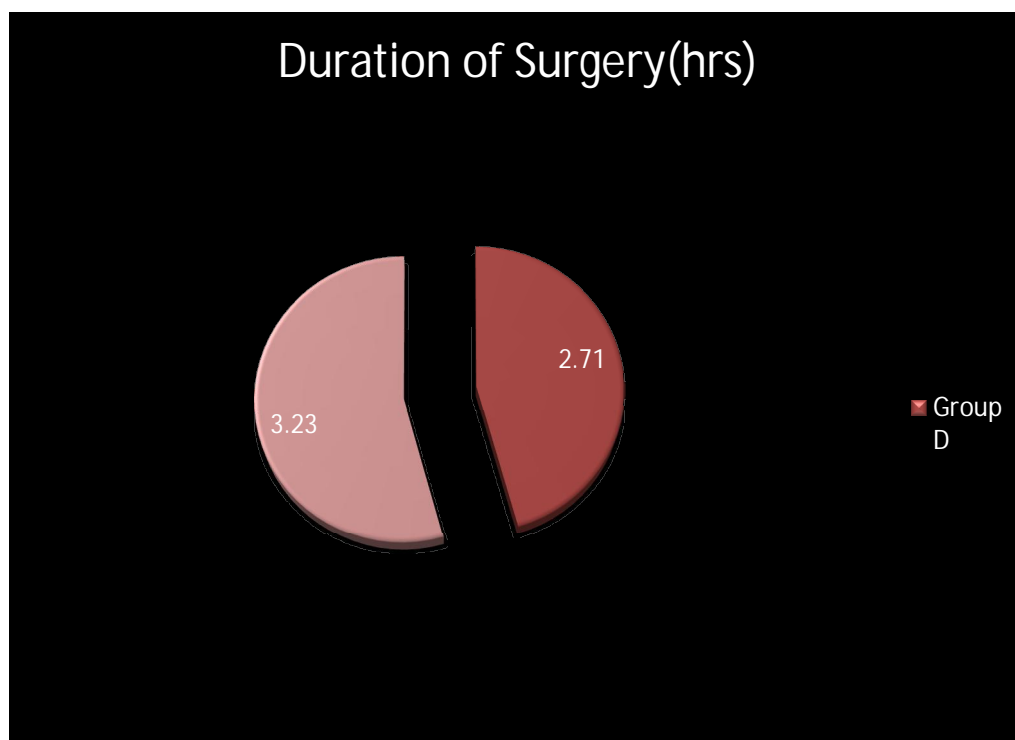
FIGURE 14

**COMPARISON OF MEAN ARTERIAL PRESSURE (MAP) OF
THE TWO GROUPS AT DIFFERENT TIME INTERVALS OF
THE STUDY**



The two groups were comparable pre operatively and at baseline with respect to pulse rate, and blood pressure (systolic, diastolic and mean arterial pressure) and there was no significant change in these parameters at 15, 30, 45 and 60 min after the block as shown in Table 3-6 and figure 10-14.

FIGURE 15
COMPARISON OF DURATION OF SURGERY BETWEEN THE TWO GROUPS



As shown in figure 15, there was no significant difference in the duration of the surgery between the two groups (3.70 ± 1.28 in group D Vs 3.23 ± 0.99 in group B, $p=0.22$)

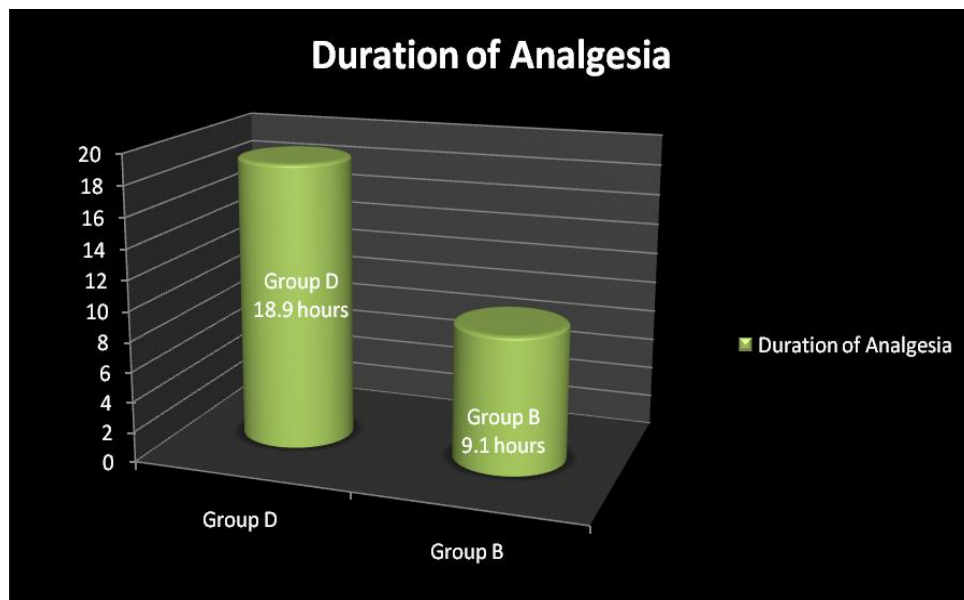
TABLE 7

**COMPARISON OF DURATION OF ANALGESIA BETWEEN
THE TWO GROUPS.**

GROUPS	DURATION OF ANALGESIA (HOURS)
GROUP D	18.97 ± 2.67
GROUP B	9.10 ± 1.79

FIGURE 16

**COMPARISON OF DURATION OF ANALGESIA BETWEEN
TWO GROUPS**



As shown in Table 7 figure 16, there was a significant prolongation in the duration of analgesia in group D as compared to group B (18.90 ± 2.67 vs 9.10± 1.79 ,p<0.001)

TABLE 8

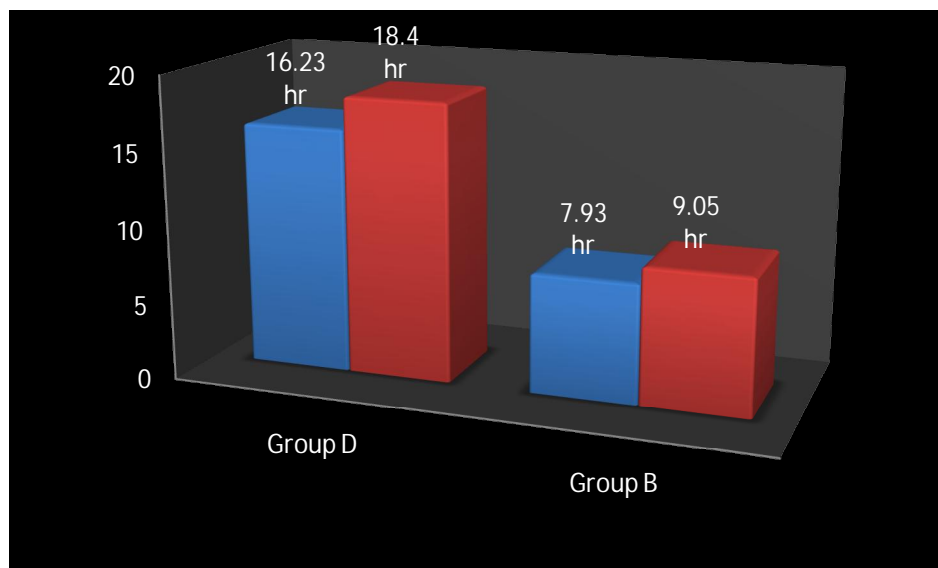
COMPARISON OF VAS SCORES OF THE TWO GROUPS

VAS scores	Group D (Mean \pm SD)	Group B (Mean \pm SD)	p value
Time to reach V2 (hrs)	16.23 \pm 2.99	7.93 \pm 1.36	<0.001*
Time to reach V4 (hrs)	18.4 \pm 2.8	9.05 \pm 1.69	<0.001*

*highly significant (p<0.001)

V2 – vas score of 2, V4 - vas score of 4.

FIGURE 17



■ V2(time to reach vas score of 2)

■ V4(time to reach vas score of 4)

As shown in table 8 and figure 17, there was a significant prolongation of the time to reach VAS score of 2 in group D as compared to group B (16.23 ± 2.99 hrs in group D vs 7.93 ± 1.36 hrs in group B, $p < 0.001$). Similarly, the time to reach VAS scores of 4 was also significantly prolonged in group D as compared to group B (18.4 ± 2.8 hrs vs 9.05 ± 1.69 hrs, $p < 0.001$). The analgesia lasted for a longer time in the dexamethasone group compared to bupivacaine group.

DISCUSSION

Eighty patients under going upper limb surgery were randomised into two groups as dexamethasone group (group D) and Bupivacaine group (group B) by slips in box method .All the baseline data were comparable between the two groups .Thus bias in our study is excluded.

In our study, we used dexamethasone as an adjuvant to local anesthetics. The primary outcomes assessed were onset of sensory and motor blockade and the duration of analgesia.

Sensory blockade in our study and control group were 4.2 ± 0.99 min and 13.6 ± 60 min respectively while the mean onset of motor blockade in group D and group B was 7.5 ± 3.2 and 11.5 ± 3.43 minutes respectively. Both these data were statistically significant($p < 0.0001$). So our study showed that there was a significant early onset of sensory and motor blockade in the study group as compared to the control group.

This results were in concordance to the findings of a study by Islam et al. who reported a significant early onset of sensory blockade (9.89 ± 1.97 min vs 11.64 ± 2.19 min, $p < 0.05$) and motor blockade (11.09 ± 2.19 minutes Vs 13.32 ± 9.8 min, $p < 0.05$) in the study group when to the compared to the control group .

In another often quoted study, Shreshtha et al found a significant early onset of sensory blockade in the steroid plus local anesthetic group as compared to the local anesthetic only group (14.50 ± 2.10 min Vs 18.15 ± 4.25 min).

They had further compared tramadol and dexamethasone as an additive to bupivacaine in supraclavicular block and showed a significant earlier onset of sensory blockade in the dexamethasone group (16.76 ± 2.34 Vs 18.47 ± 2.03 , $p=0.004$) . This early onset of blockade with dexamethasone may be due to synergistic action with local anesthetic drug in blocking the nerve fibres.

The duration of analgesia is one of the primary outcomes that has been studied in all these studies where dexamethasone has been studied as an adjuvant to local anesthetics. In the study by Shreshtha et al comparing the analgesic efficacy of dexamethasone with tramadol, the duration of analgesia had significantly prolonged in dexamethasone group (1028.17 ± 194.51 min vs 453.17 ± 72.87 min, $p<0.001$) .

Yadav et al in a double blinded study studied the effectiveness of adding dexamethasone or neostigmine to local anesthetic and showed that the duration of analgesia was maximum in the dexamethasone group (454.2 ± 110.7 Vs 225.7 ± 53.3 Vs 176.5 ± 53.5 , $p<0.001$) .

Parrington et al in a randomised controlled study found that addition of dexamethasone 8 mg to mepivacaine in supraclavicular block dramatically increased the mean duration of analgesia to about 332 minutes as compared to 228 min in the control group. Islam et al also showed similar prolongation in the duration of analgesia on addition of dexamethasone to local anesthesia (11.87 ± 0.53 vs 3.43 ± 0.49 , $p < 0.001$)

Pathak et al did a comparative study on supraclavicular block with or without dexamethasone to local anesthetic and showed that the duration of analgesia was prolonged to 834 ± 78.1 in the study group as compared to 276 ± 38.73 minutes in the control group.

Our study was in concordance with all these previous studies in the fact that the analgesia duration was significantly prolonged in the study group to 18.90 ± 2.67 min as compared to 9.10 ± 1.79 min in the control group. ($p < 0.001$).

During this study, two patients from the study group showed 24 hours of analgesia which may not be statistically significant, but shows some clinical relevance to investigate further.

SUMMARY

Regional anesthesia is preferred to general anesthesia for upper limb procedures since they provide excellent analgesia which extends into the postoperative period.

This study compared the analgesic efficacy of dexamethasone as an adjuvant to bupivacaine in supraclavicular brachial plexus block. This is a prospective randomised controlled study. In this study, eighty patients were randomly divided into two groups as group D (Study group) and group B (control group). The study group received 25 ml of 0.5% bupivacaine with 1.25ml of 1 in 10000 epinephrine and 8mg of dexamethasone. The control group received 25 ml of 0.5% bupivacaine with 1.25ml of 1 in 10000 epinephrine. Total volume of the local anesthetic was made to 28 ml by adding distilled water.

Epinephrine was added to decrease systemic absorption and to reduce local anesthetic toxicity. Supraclavicular block given with nerve stimulator technique by classical approach. Onset of motor and sensory block and duration of analgesia were observed. In this study, mean onset of motor block were 7.5 ± 3.2 minutes in the dexamethasone group and 11.5 ± 3.43 minutes in the Bupivacaine group respectively.

The mean onset of sensory block was 4.2 ± 0.99 minutes in the dexamethasone group and 13.6 ± 6 minutes in the bupivacaine group. Both the results show early onset of motor and sensory block in the study group which was statistically significant ($p < 0.0001$). Mean duration of analgesia was 18.9 ± 2.68 in the study group. The duration of analgesia was significantly longer and VAS scores were lower in the dexamethasone group compared to the Bupivacaine group.

CONCLUSION

From this study it is concluded that,

Dexamethasone in the dose of 8mg given as an adjuvant in supraclavicular block along with Bupivacaine and epinephrine results in early onset of motor and sensory block . The duration of analgesia is also significantly prolonged. The prolonged postoperative pain relief helps in early mobilisation and thereby improving physical and psychological wellbeing of the patient .

This dose is optimal in that it does not cause any side effects at the same time it provides excellent intraoperative and postoperative analgesia.

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PATIENT CONSENT FORM

Study title

A prospective Randomized controlled study comparing the Analgesic efficacy of Dexamethasone when added to Bupivacaine in supraclavicular brachial plexus block for upper limb surgeries .

Study centre : ESI – PGIMSR, K.K.NAGAR, CHENNAI -78

Participant name :

Age:

Sex:

I confirm that I have understood the purpose of procedure for the above study . I have the opportunity to ask the question and all my questions and doubts have been answered to my satisfaction.

I have been explained about the pitfall in the procedure. I have been explained about the safety, advantage and disadvantage of the technique. I understand that my participation in the study is voluntary and that I am free to withdraw at anytime without giving any reason.

I understand that investigator ,regulatory authorities and the ethics committee will not need my permission to look at my health records both in respect to current study and any further research that may be conducted in relation to it, even if I withdraw from the study . I understand that my identity will not be revealed in any information released to third parties or published , unless as required under the law . I agree not to restrict the use of any data or results that arise from the study.

I understand that that I will receive drugs to prolong the duration of analgesia using dexamethasone in supraclavicular brachial plexus block. I have been explained that the anesthetic technique is a standard and approved technique. This may help in future research in the field of anesthesia. I consent to undergo this procedure

Insurance No:

Date:

Signature / thumb impression of

PROFORMA

Name of the patient:

Age:

Sex:

Wt:

Insurance No:

OT:

Diagnosis:

Duration of Procedure:

Surgeon:

Anaesthetist

PREOPERATIVE DETAILS

ASA Grade

Remarks:

vitals

BP	Pulse rate	Resp. rate	SpO2	Temp	ECG	Xray

Hb	RBS	RFT	LFT	Others

PROFORMA

BASELINE DATA	GROUP - D	GROUP -B	P VALUE
AGE			
SEX			
WEIGHT			
HEIGHT			
BMI			
LEVEL OF SURGERY I-elbow and above elbow II –Forearm III - Hand			
TYPE OF SURGERY I- Elective II- Emergency			
ASA I II			

INTRAOPERATIVE DETAILS:

EXPERIMENTAL PARAMETERS

	GROUP D	GROUP B	P VALUE
Onset of sensory blockade			
Onset of motor blockade			
Duration of Analgesia			

VITAL PARAMETERS:

1.PULSE RATE

Groups	Pre op	0 min	15 min	30 min	45 min	60min	P value
Group D							
Group B							

2.SYSTOLIC BP

Groups	Pre op	0 min	15 min	30 min	45 min	60 min	P value
Group D							
Group B							

3.DIASTOLIC BP

Groups	Pre op	0 min	15 min	30 min	45 min	60 min	P value
Group D							
Group B							

4. MEAN ARTERIAL PRESSURE

Groups	Preop	0 min	15min	30 min	45 min	60 min	P values
Group D							
Group B							

POSTOPERATIVE PERIOD

VAS SCORES

GROUPS	1 st HR	2 nd HR	3 rd HR	4 th HR	5 th HR
GROUP D					
GROUP B					

GROUPS	6 th HR	7 th HR	8 th HR	9 th HR	10 th HR
GROUP D					
GROUP B					

GROUPS	12 th HR	14 th HR	16 th HR	18 th HR	20 th HR	22 th HR	24 th HR
GROUP D							
GROUP B							

SIGNATURE OF INVESTIGATOR
PARTICIPANT

SIGNATURE OF THE
WITNESS

NAME	GROUP	AGE	SEX	IP NO	WEIGHT	HEIGHT	BMI	ASA	OF	SUD	SUF	PR-pr	SYS-pr	DIA-pr	MAP-pr	SPO2-pr	ON_MB	ON_SB	PR-0	SYS-0	DIA-0	MAP-0	SPO2-0	PR-15	SYS-15	DIA-15	MAP-15
mohanaraman	D	47	M	51-11933469	78	172	26.3656	I	II	I	I	88	138	80	99.3	100	5	4	72	130	80	96.7	99	72	133	74	93.7
Manikandan	B	21	M	51-60474674	71	169	24.85907	I	II	I	I	110	124	78	93.3333333	100	15	12	104	124	75	91.3333333	100	104	122	72	88.667
Alaguraja	D	22	M	51-21870502	54	160	21.09375	II	II	I	I	98	138	88	105	99	5	4	90	132	80	97.3	100	90	130	80	96.7
Balakumar	D	60	M	51-21605169	69	170	23.87543	II	II	II	I	78	102	70	80.7	100	5	4	86	106	70	82	100	82	112	72	85.3
Nagarajan	B	41	M	51-20451200	78	176	25.18079	I	II	I	I	106	144	96	112	100	15	18	104	140	74	96	100	102	142	72	95.333
Kokila	D	37	F	51-22836072	54	156	22.18935	I	I	I	I	102	132	72	92	100	5	4	92	128	68	87.3	100	90	131	74	93
Prabu	B	33	M	51-22295067	76	175	24.81633	I	I	I	I	108	134	70	91.3333333	100	10	12	107	132	70	90.6666667	100	105	130	70	90
Bupesh	B	29	M	51-22647388	64	157	25.96454	I	II	I	I	88	108	76	86.6666667	100	10	18	92	112	78	89.3333333	100	94	106	74	84.667
Kannian	D	40	M	51-15225102	68	168	24.09297	II	I	I	I	88	136	76	96	99	5	4	80	130	70	90	100	82	130	80	96.7
Annadurai	B	45	M	51-13435637	76	174	25.10239	II	I	I	I	104	150	94	112.666667	100	10	14	102	142	98	112.666667	100	100	148	90	109.33
Natarajan	B	53	M	51-22374248	69	176	22.27531	II	I	I	I	102	100	70	80	100	10	16	100	102	70	80.6666667	100	92	104	68	80
Manoj kumar	D	19	M	51-22112248	58	162	22.10029	I	II	I	I	86	128	68	88	100	5	4	84	126	74	91.3	100	80	124	72	89.3
Anthoniamma	D	26	F	51-1661216	58	162	22.10029	I	II	I	I	90	109	68	81.7	100	10	4	92	112	72	85.3	100	82	104	74	84
Jeyaraman	B	49	M	51-22586063	75	179	23.40751	I	II	I	I	92	110	72	84.6666667	100	15	10	96	112	72	85.3333333	100	94	114	72	86
Selvam	D	42	M	51-16684712	60	172	20.28123	I	III	I	I	98	126	86	99	99	10	4	86	120	82	94.7	100	78	120	78	92
Neela	B	60	F	51-13255005	62	165	22.77319	II	I	I	I	82	108	74	85.3333333	100	15	18	88	109	74	85.6666667	100	82	104	72	82.667
Kokila	B	37	F	51-22072836	66	169	23.10843	I	I	I	I	84	124	72	89.3333333	100	5	14	82	128	90	102.666667	100	86	126	70	88.667
Jaya	D	32	F	51-2184650	58	158	23.23346	I	III	II	I	104	142	76	98	100	10	2	90	138	72	94	100	92	136	72	93.3
Raji	B	25	M	51-15362810	66	172	22.30936	I	III	II	I	76	132	70	90.6666667	100	10	16	78	130	72	91.3333333	100	78	128	78	94.667
Jayalakshmi	B	58	F	51-13150201	64	164	23.79536	II	II	I	I	98	120	70	86.6666667	100	10	12	94	122	74	90	100	102	124	72	89.333
Arumugam	D	29	M	51-17157715	64	164	23.79536	I	I	I	I	92	132	72	92	100	10	4	90	132	70	90.7	100	92	131	72	91.7
Sathish kumar	B	42	M	51-14408541	70	168	24.80159	II	II	I	I	78	124	74	90.6666667	100	15	10	88	132	76	94.6666667	100	102	134	70	91.333
Durai raj	D	53	M	51-17396017	70	174	23.12062	II	II	I	I	102	146	76	99.3	99	10	4	100	140	72	94.7	99	88	140	70	93.3
Abdul kadher	B	29	M	51-17295652	71	169	24.85907	I	II	I	I	84	122	70	87.3333333	100	15	20	92	140	90	106.666667	100	88	132	70	90.667
Sadagopan	B	49	M	51-22810291	65	162	24.76757	I	II	I	I	90	112	78	89.3333333	100	5	12	102	120	72	88	100	90	114	72	86
Vasanthi	D	48	F	51-16708888	58	162	22.10029	II	I	I	I	98	132	76	94.7	99	5	6	78	130	72	91.3	99	72	126	78	94
Balasubramaniam	B	18	M	51-15373629	72	165	26.44628	I	II	I	I	92	104	74	84	100	10	18	88	114	74	87.3333333	100	86	124	70	88
Priya ranjan d	D	28	F	51-21105433	52	158	20.83	I	II	I	I	88	112	68	82.7	100	10	6	80	110	70	83.3	100	82	108	74	85.3
Shankar	B	45	M	51-16682279	62	159	24.52435	II	III	II	I	68	104	78	86.6666667	100	15	12	76	108	72	84	100	82	110	70	83.333
Paraimala	B	44	F	51-20504311	79	176	25.50362	II	I	I	I	96	104	62	76	100	10	8	102	108	74	85.3333333	100	98	106	72	83.333
Umesh raj	B	28	M	51-22503174	68	167	24.38237	I	II	I	I	72	124	74	90.6666667	100	10	8	82	120	70	86.6666667	100	88	126	74	91.333
manoj kumar	D	51	M	51-17773028	79	165	29.01745	II	II	I	I	84	140	98	112	99	10	2	82	132	78	96	100	82	130	72	91.3
manoharan	D	54	M	51-66323018	72	171	24.62296	II	II	I	I	98	102	62	75.3	100	10	4	92	110	70	83.3	100	84	122	74	90
Albert robinson	D	25	M	51-17614602	66	162	25.14861	I	II	II	I	89	100	70	80	100	10	4	74	102	76	84.7	100	76	104	74	84
Johnwestley	B	21	M	51-17551091	72	172	24.33748	I	II	I	I	78	98	70	79.3333333	100	15	10	88	100	72	81.3333333	100	82	100	72	81.333
Mahendran	D	32	M	51-15622744	69	168	24.44728	I	I	I	I	106	136	86	102.7	99	10	4	82	132	78	96	100	78	132	72	92

Karthikeyan	D	24	M	51-21963256	67	168	23.73866	I	II	I	88	120	74	89.3	100	5	6	98	118	74	88.7	100	92	118	64	82
Arumugam	B	31	M	51-21367524	74	176	23.88946	I	I	I	104	132	70	90.6666667	100	5	12	92	134	70	91.3333333	100	90	130	72	91.333
Vigneswaran	D	23	M	51-20880248	70	169	24.50895	I	II	I	108	120	70	86.7	100	5	4	88	118	72	87.3	100	82	116	78	90.7
Thanikesan	D	58	M	51-17430796	76	178	23.98687	II	II	I	86	120	82	94.7	100	10	4	86	130	80	96.7	100	84	122	78	91.3
Naresh Rai	B	34	M	51-20422716	69	174	22.79033	I	I	I	108	130	90	103.333333	100	15	12	102	128	76	93.3333333	100	92	120	70	86.667
Babu	D	27	M	51-2245288	69	172	23.32342	I	III	II	92	132	70	90.7	100	5	6	92	118	74	88.7	100	84	124	72	89.3
Senthil	D	31	M	51-17247114	70	171	23.93899	I	I	I	96	130	84	99.3	99	10	4	88	128	80	96	100	86	122	80	94
Kalaiarasan	B	23	M	51-20030920	69	170	23.87543	I	II	I	98	140	90	106.666667	100	15	14	104	132	70	90.6666667	100	98	130	72	91.333
Sathish	B	33	M	51-11166707	80	177	25.53545	I	II	I	106	122	72	88.6666667	100	15	16	98	132	70	90.6666667	100	99	132	82	98.667
Prabakaran	D	21	M	51-162 46180	62	168	21.96712	I	II	I	112	110	72	83.3	100	15	4	109	132	70	90.7	98	102	114	70	84.7
Murugan	D	38	M	51-13349928	64	163	24.08822	I	I	I	98	104	72	82.7	100	5	4	102	108	72	84	100	98	126	70	88.7
Paneerselvam	B	48	M	51-21708635	72	168	25.5102	I	I	I	84	132	70	90.6666667	100	15	18	92	138	80	99.3333333	100	92	126	78	94
Isravel	D	42	M	51-16258990	68	164	25.28257	I	III	I	106	122	70	87.3	100	5	4	102	120	82	94.7	100	98	118	70	86
Khokanmajun	B	21	M	51-12352922	65	169	22.75831	I	II	I	82	144	74	97.3333333	100	10	20	90	130	90	103.333333	100	102	136	70	92
Mohan	B	21	M	51-23118538	63	157	25.55885	I	III	I	86	118	72	87.3333333	100	10	12	90	128	70	89.3333333	100	88	132	70	90.667
Muthu velayud	D	29	M	51-22080634	67	178	21.14632	I	I	I	78	120	74	89.3	100	5	6	84	118	72	87.3	100	82	120	60	80
Palani	B	40	M	51-20468677	70	168	24.80159	II	II	I	102	112	72	85.3333333	100	10	10	104	122	90	100.666667	100	98	128	70	89.333
Gnanasekara	B	25	M	51-23188203	69	174	22.79033	I	II	I	98	108	68	81.3333333	100	10	20	102	106	74	84.6666667	100	98	110	74	86
Dharmarajan	D	32	M	51-20797346	69	165	25.34435	I	II	I	108	118	76	90	100	5	4	108	118	72	87.3	100	106	116	74	88
Natarajan	D	28	M	51-15812875	64	164	23.79536	I	I	I	92	114	73	86.7	100	5	2	88	102	72	82	100	82	108	72	84
Narayanan	B	51	M	51-17342822	71	168	25.1559	II	II	I	88	128	70	89.3333333	100	10	12	92	128	70	89.3333333	100	98	124	76	92
Velmurugan	D	27	M	51-15922802	66	164	24.53896	I	III	I	102	120	72	88	100	5	4	92	122	68	86	100	90	124	62	82.7
Babu	D	32	M	51-1582857	64	159	25.31545	I	III	I	102	100	72	81.3333333	100	5	4	90	112	70	84	100	82	109	74	85.667
Senthamizh	B	37	F	51-14449595	62	155	25.80645	I	II	I	82	112	68	82.6666667	100	15	8	98	118	70	86	100	102	116	72	86.667
Arumugam	D	53	M	51-17636642	69	166	25.03992	II	I	I	94	114	64	80.7	100	10	4	94	128	64	85.3	100	92	124	72	89.3
Ramadoss	B	24	M	51-15819139	65	158	26.03749	I	II	I	98	122	70	87.3333333	100	5	14	98	126	72	90	100	102	124	72	89.333
Ramesh	D	23	M	51-21410823	72	170	24.91349	I	III	II	96	112	68	82.7	100	15	6	96	108	72	84	100	94	108	71	83.3
Nirmala	B	40	F	51-14416972	70	166	25.40282	II	II	II	98	104	74	84	100	5	12	98	116	72	86.6666667	100	98	122	70	87.333
Udayakumar	D	24	M	51-21652667	76	174	25.10239	I	III	II	88	108	78	88	100	5	4	86	108	70	82.7	100	84	106	72	83.3
Govindhasam	B	50	M	51-21224619	76	178	23.98687	II	I	I	104	112	72	85.3333333	100	10	14	98	122	76	91.3333333	100	92	112	74	86.667
praburaj	D	29	M	51-16840208	59	164	21.93635	I	III	II	84	107	76	86.3	100	10	4	86	107	74	85	100	84	108	70	82.7
Jeyaraj	D	28	M	51-22506162	62	164	23.05175	I	I	I	76	110	72	84.7	100	5	6	86	112	70	84	100	88	122	76	91.3
Babu	D	34	M	51-13810418	72	173	24.05693	I	II	I	86	106	70	82	100	5	4	84	106	70	82	100	82	128	74	92
Ramesh	D	48	M	51-13512946	60	159	23.73324	II	III	II	104	112	70	84	100	15	4	102	108	72	84	100	106	112	72	85.3
Anbukannan	B	35	M	51-22098288	65	156	26.7094	II	II	I	72	108	72	84	100	10	20	88	120	70	86.6666667	100	82	134	72	92.667
Mani	B	48	M	51-22924421	64	159	25.31545	I	III	I	108	114	62	79.3333333	99	10	12	102	118	76	90	100	98	120	76	90.667
Sabarish	D	18	M	51-16652909	59	158	23.63403	I	II	I	98	118	76	90	100	5	4	98	124	74	90.7	100	98	122	72	88.7

Venkatesh	B	51	M	51-15717675	64	156	26.29849	I	II	I	98	108	70	82.6666667	100	10	10	102	110	72	84.6666667	100	98	114	74	87.333
vijayalakshmi	D	32	M	51-13850364	61	162	23.24341	I	II	I	92	109	62	77.7	100	5	4	108	108	64	78.7	99	107	108	62	77.3
kalaimani	B	31	M	51-23347731	65	162	24.76757	I	II	I	92	104	68	80	100	15	12	102	112	70	84	100	100	118		#####
Harikrishnan	D	38	M	51-13069013	62	161	23.91883	I	II	I	89	100	76	84	100	5	4	102	103	70	81	100	91	105	74	84.3
Thangasamy	B	28	M	51-23543781	68	154	28.67263	I	II	I	88	110	70	83.3333333	100	15	14	92	116	72	86.6666667	100	98	124	78	93.333
Sasikala	B	53	F	51-17652629	50	148	22.82688	II	III	I	78	124	80	94.6666667	100	15	10	96	130	72	91.3333333	100	92	130	70	90
Mohanrajan	B	47	M	51-11933469	66	154	27.82931	II	II	I	94	106	78	87.3333333	100	15	15	102	112	78	89.3333333	100	98	120	76	90.667

SPO2-15	PR-30	SYS-30	DIA-30	MAP-30	SPO2-30	PR_45	SYS_45	DIA_45	MAP_45	PO2_45	PR_60	SYS-60	DIA_60	MAP_60	IR_DURATION	P_1	P_2	P_3	P_4	P_5	P_6	P_7	P_8	P_9	P_10	P_12	P-14	P-16	P-18	P_20	P_22	P-24	
99	76	127	78	94	99	74	126	76	92.6666667	99	74	122	72	88.6666667	3	18				VO	VO	VO	VO	VO	VO	VO	VO	VO	V2	V4			
100	102	120	79	92.6667	100	98	124	74	90.6666667	100	98	122	72	88.6666667	5	12						VO	VO	V2	V2	V4							
100	90	128	74	92	100	84	122	74	90	100	82	128	72	90.6666667	3	16				VO	VO	VO	VO	VO	VO	VO	VO	V2	V4				
100	84	122	70	87	100	80	124	72	89.3333333	100	76	118	70	86	3.3	18				VO	VO	VO	VO	VO	VO	VO	VO	VO	V2	V4			
100	104	140	72	94.6667	100	98	132	90	104	100	98	130	70	90	4.3	8					VO	V2	V2	V4									
100	86	129	72	91	100	86	128	72	90.6666667	100	72	124	70	88	5	16					VO	VO	VO	VO	VO	VO	V2	V4					
100	102	128	74	92	100	100	126	76	92.6666667	100	102	130	72	91.3333333	4	9					VO	VO	V2	V4									
100	98	120	72	88	100	82	108	72	84	100	92	106	74	84.6666667	4	10					VO	VO	VO	VO	V2	V4							
100	84	128	72	91	100	88	122	80	94	100	88	122	80	94	1	14	VO	VO	VO	VO	VO	VO	VO	VO	VO	V2	V4						
100	98	148	70	96	100	102	146	84	104.666667	100	94	142	72	95.3333333	4	10					VO	VO	V4										
100	104	108	72	84	100	96	108	62	77.3333333	100	102	106	70	82	3	7					VO	V2	V4										
100	78	122	72	89	100	78	120	72	88	100	78	120	70	86.6666667	2	22			VO	VO	VO	VO	VO	VO	VO	VO	VO	VO	VO	VO	V2	V4	
100	84	102	78	86	100	92	108	78	88	100	94	109	72	84.3333333	3	20				VO	VO	VO	VO	VO	VO	VO	VO	VO	VO	VO	V4		
100	92	116	74	88	100	94	112	72	85.3333333	100	94	110	72	84.6666667	4	6					VO	V4											
100	76	122	71	92	100	74	122	70	87	100	76	122	70	87.3333333	4	18					VO	VO	VO	VO	VO	VO	V2	V2	V2	V4			
100	86	106	72	83.3333	100	84	104	70	81.3333333	100	92	102	68	79.3333333	3	8				VO	VO	VO	VO	V4									
100	84	124	70	88	100	82	122	72	88.6666667	100	84	120	70	86.6666667	2.4	9					VO	VO	V2	V2	V4								
100	86	142	74	97	100	78	142	74	96.6666667	100	76	138	70	92.6666667	3	12				VO	VO	VO	VO	VO	VO	VO	V4						
100	82	126	72	90	100	84	120	70	86.6666667	100	82	114	76	88.6666667	3	12				VO	VO	VO	VO	V2	V4								
100	94	124	68	86.6667	100	96	120	72	88	100	94	118	76	90	3.3	8					VO	V2	V4										
100	78	126	70	89	100	74	128	70	89.3333333	100	74	124	68	86.6666667	1.2	16	VO	VO	VO	VO	VO	VO	VO	VO	VO	VO	V2	V4					
100	82	128	78	94.6667	100	102	118	70	86	100	78	116	74	88	4	10					VO	VO	VO	VO	V2	V4							
99	76	138	71	93	99	76	145	72	96.3333333	99	76	136	70	92	2	16			VO	VO	VO	VO	VO	VO	VO	VO	VO	VO	V2	V4			
100	92	120	90	100	100	82	116	70	85.3333333	100	84	107	90	95.6666667	3	6				VO	VO	V2	V4										
100	104	104	74	84	100	84	112	70	84	100	94	114	72	86	3	9				VO	VO	VO	VO	V2	V4								
100	74	122	74	90	100	80	122	74	90	100	80	118	74	88.6666667	2	18			VO	VO	VO	VO	VO	VO	VO	VO	V2	V2	V4				
100	102	110	78	88.6667	100	92	118	74	88.6666667	100	86	110	84	92.6666667	3.3	9					VO	VO	VO	V2	V4								
100	80	106	72	83	100	84	144	78	100	100	84	139	74	95.6666667	3	18				VO	VO	VO	VO	VO	VO	VO	VO	VO	VO	V4			
100	90	114	78	90	100	86	120	70	86.6666667	100	74	110	70	83.3333333	1	12		VO	VO	VO	VO	VO	VO	VO	VO	V2	V4						
100	90	108	72	84	100	98	98	72	80.6666667	100	82	104	82	89.3333333	3	10				VO	VO	VO	VO	VO	V2	V4							
100	86	120	70	86.6667	100	90	118	72	87.3333333	100	78	112	74	86.6666667	4	6					VO	VO	V4										
100	82	126	70	89	100	82	126	72	90	100	82	122	74	90	4	18					VO	VO	VO	VO	VO	VO	V2	V2	V4				
100	78	120	72	88	100	86	122	74	90	100	82	120	72	88	3	22				VO	VO	VO	VO	VO	VO	VO	VO	VO	VO	V2	V4		
100	76	105	72	83	100	76	114	72	86	100	70	108	70	82.6666667	4	24					VO	VO	VO	VO	VO	VO	VO	VO	VO	VO	VO	V2	V4
100	84	114	72	86	100	80	112	70	84	100	80	108	72	84	1.3	8			VO	VO	VO	VO	VO	V4									
100	74	131	70	90	100	76	130	80	96.6666667	100	74	124	76	92	3	20				VO	VO	VO	VO	VO	VO	VO	VO	VO	VO	V2	V4		

100	86	120	72	88	100	84	120	70	86.6666667	100	76	112	72	85.3333333	2.3	20			VO	VO	VO	VO	VO	VO	VO	VO	VO	VO	V2	V4	V0			
100	78	128	70	89.3333	100	84	134	78	96.6666667	100	98	124	74	90.6666667	2	10			VO	VO	VO	VO	VO	VO	V2	V4								
100	76	114	72	87	100	74	126	68	87.3333333	100	72	112	64	80	3	18				VO	VO	VO	VO	VO	VO	VO	VO	VO	V2	V4				
100	84	122	76	91	100	84	120	76	90.6666667	100	84	120	72	88	2	16			VO	VO	VO	VO	VO	VO	VO	VO	VO	VO	V4					
100	82	112	72	85.3333	100	94	120	72	88	100	102	114	70	84.6666667	4.4	8					VO	VO	VO	VO	V2	V4								
100	86	112	74	87	100	94	114	72	86	100	92	112	70	84	2	18			VO	VO	VO	VO	VO	VO	VO	VO	V2	V4						
100	84	120	80	93	100	82	124	82	96	100	82	120	80	93.3333333	4	20					VO	VO	VO	VO	VO	VO	VO	VO	VO	V2	V4			
100	98	128	72	90.6667	100	98	124	70	88	100	92	132	72	92	3	8				VO														
100	98	136	70	92	100	88	130	72	91.3333333	100	88	120	70	86.6666667	3	10				VO	VO	VO	VO	VO	V2	V4								
100	98	112	70	84	100	96	132	70	90.6666667	100	82	134	78	96.6666667	3	22				VO	VO	VO	VO	VO	VO	VO	VO	VO	VO	VO	VO	V4		
100	96	118	70	86	100	92	116	70	85.3333333	100	90	108	74	85.3333333	4	20					VO	VO	VO	VO	VO	VO	VO	VO	VO	V2	V4			
100	94	114	70	84.6667	100	98	120	70	86.6666667	100	102	126	74	91.3333333	3	9				VO	VO	VO	VO	V2	V4									
100	96	116	70	85	100	94	114	72	86	100	84	110	70	83.3333333	1	20			VO	VO	VO	VO	VO	VO	VO	VO	VO	VO	VO	VO	VO	V4		
100	98	128	74	92	100	98	122	72	88.6666667	100	98	118	72	87.3333333	4.3	8					VO	VO	V2	V4										
100	92	126	72	90	100	92	122	72	88.6666667	100	86	128	70	89.3333333	1.1	7			VO	VO	VO	VO	VO	V4										
100	78	118	72	87	100	76	120	72	88	100	78	116	70	85.3333333	4	14					VO	VO	VO	VO	VO	VO	V2	V4						
100	102	124	76	92	100	102	118	72	87.3333333	100	98	102	78	86	3.4	9				VO	VO	VO	VO	V2	V4									
100	98	112	72	85.3333	100	92	106	70	82	100	90	108	76	86.6666667	3	9				VO	VO	VO	VO	V0	V4									
100	105	118	74	89	100	106	118	74	88.6666667	100	98	116	74	88	2.2	18			VO	VO	VO	VO	VO	VO	VO	VO	VO	VO	V2	V4				
99	75	102	74	83	99	78	108	74	85.3333333	100	72	104	72	82.6666667	4.3	20						VO	VO	VO	VO	VO	VO	VO	V2	V2	V4			
100	102	122	76	91.3333	100	94	118	72	87.3333333	100	92	110	72	84.6666667	3.3	12					VO	VO	VO	VO	VO	VO	V4							
100	92	116	78	91	100	94	118	76	90	100	88	114	74	87.3333333	1	22			VO	VO	VO	VO	VO	VO	VO	VO	VO	VO	VO	V2	V4			
100	78	108	78	88	99	82	102	72	82	100	82	100	74	82.6666667	3	18						VO	VO	VO	VO	VO	VO	V2	V2	V4				
100	98	122	70	87.3333	100	96	120	70	86.6666667	100	96	114	64	80.6666667	4	8					VO	VO	V2	V4										
100	90	121	76	91	100	91	122	74	90	100	89	120	74	89.3333333	4	20				VO	VO	VO	VO	VO	VO	VO	VO	VO	VO	V4				
100	98	118	72	87.3333	100	98	120	68	85.3333333	100	102	118	72	87.3333333	4	10					VO	VO	VO	VO	V2	V4								
100	94	106	62	77	100	92	107	64	78.3333333	100	96	102	74	83.3333333	1.3	20			VO	VO	VO	VO	VO	VO	VO	VO	VO	VO	VO	VO	V2	V4		
100	102	130	72	91.3333	100	96	128	82	97.3333333	100	98	120	64	82.6666667	3	10				VO	VO	VO	VO	VO	V2	V4								
100	86	102	72	82	100	84	104	68	80	100	82	102	60	74	2	22				VO	VO	VO	VO	VO	VO	VO	VO	VO	VO	VO	V4			
100	98	102	70	80.6667	100	94	100	62	74.6666667	100	90	104	72	82.6666667	4	8						VO	VO	V2	V4									
100	88	110	70	83	100	86	130	72	91.3333333	100	82	127	72	90.3333333	1.3	22			VO	VO	VO	VO	VO	VO	VO	VO	VO	VO	VO	V2	V4			
100	86	126	74	91	100	84	124	74	90.6666667	100	82	116	72	86.6666667	2	18				VO	VO	VO	VO	VO	VO	VO	VO	V2	V4					
100	84	128	74	92	100	86	131	72	91.6666667	100	84	129	72	91	2.3	18				VO	VO	VO	VO	VO	VO	VO	VO	VO	V2	V4				
100	98	110	72	85	100	96	132	72	92	100	98	128	74	92	1.3	22					VO	VO	VO	VO	VO	VO	VO	VO	VO	V2	V4			
100	84	130	72	91.3333	100	82	132	72	92	100	84	124	72	89.3333333	3	12				VO	VO	VO	VO	VO	V2	V4								
100	98	122	70	87.3333	100	102	118	72	87.3333333	100	98	114	72	86	4	12						VO	VO	VO	VO	V2	V2	V4						
100	96	122	72	89	100	94	121	76	91	100	97	120	68	85.3333333	1	18			VO	VO	VO	VO	VO	VO	VO	VO	VO	V2	V4					

100	98	120	68	85.3333	100	98	118	70	86	100	96	108	72	84	2	9				VO	VO	VO	VO	VO	V2	V4								
100	102	102	62	75	100	98	104	68	80	100	98	106	62	76.6666667	3	20				VO	VO	VO	VO	VO	VO	VO	VO	VO	V2	V2	V4			
100	98	118	72	87.3333	100	104	114	72	86	100	94	120	70	86.6666667	3	6				VO	V2	V4												
100	88	102	72	82	100	92	105	72	83	100	82	102	70	80.6666667	5	24						VO	VO	VO	VO	VO	VO	VO	VO	VO	VO	VO	V2	V4
100	96	120	76	90.6667	100	98	116	75	88.6666667	100	92	122	74	90	5	10						VO	VO	VO	VO	V4								
100	90	128	70	89.3333	100	90	130	74	92.6666667	100	84	122	70	87.3333333	1	8		VO	VO	VO	VO	V2	V4											
100	102	124	74	90.6667	100	98	122	76	91.3333333	100	92	120	72	88	3	12				VO	V0	VO	VO	VO	VO	VO	V2	V4						

ஒப்புதல் படிவம்

1. எனக்கு.....அறுவை சிகிச்சையை செய்யுமாறு நுளுஐஊ மருத்துவர் மற்றும் குழுவினரை வேண்டிக் கொள்கிறேன்.
2. நோயின் தன்மை :
சிகிச்சை முறை :
இவை அனைத்தும் எனக்கு மருத்துவர் மூலம் தெளிவாக விளக்கப்பட்டன.
3. எனக்கு கழுத்தில் மரத்துப் போகிற ஊசி போட்டு கையை மரத்துப் போக செய்ய ஒப்புக்கொள்கிறேன். இதற்கு உபயோகப்படுத்தப்படும் மருந்துகளைப் பற்றி மருத்துவர் மூலம் அறிந்து கொண்டேன்.
4. இவற்றின் பின்விளைவுகளை மருத்துவர் மூலம் அறிந்துக் கொண்டேன்.
5. அனைத்து மருத்துவ சிகிச்சை முறைகளின் நிறைகளும் குறைகளும் எனக்கு விளக்கப்பட்டன.
6. மேலே கொடுக்கப்பட்டுள்ள அனைத்தும் மருத்துவமனை நன்னெறி (Ethics) குழுவின் வரைமுறைகளுக்கு உட்பட்டே நடக்கும் என மருத்துவர் விளக்கினார். மேலும் இந்த சிகிச்சை முறைகளுக்கு உடன்பட மறுக்கவும் எனக்கு உரிமை உண்டு என்பதை நான் அறிவேன்.
7. என் சிகிச்சையின் போது கிடைக்கும் தகவல்களை மருத்துவ ஆராய்ச்சிக்கு பயன்படுத்தவும் சம்மதம் அளிக்கிறேன்

நான் இந்த ஒப்புதல் படிவத்தை படித்த பின்னரே / படித்துக் காண்பிக்கப்பட்ட பின்னரே இதன் சாராம்சத்தை முழுவதுமாக புரிந்துக் கொண்டு பின்பே முழுமனதுடன் சம்மதித்து கையெழுத்திடுகிறேன்.

சாட்சி :

ஒப்புதல் அளிப்பவர்

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KEY TO MASTER CHART

GROUPS

D (STUDY) ---- DEXAMETHASONE GROUP

B (CONTROL)----- BUPIVACAINE GROUP

LEVEL OF SURGERY

I - ELBOW AND ABOVE ELBOW SURGERIES.

II - FOREARM SURGERIES

III - HAND SURGERIES.

TYPE OF SURGERY

I - ELECTIVE

II - EMERGENCY

ON_SB – ONSET OF SENSORY BLOCK.

ON_MB – ONSET OF MOTOR BLOCK.

DUR_SUR - DURATION OF SURGERY.

DUR_AN - DURATION OF ANALGESIA.

P_1 TO P_24 - Ist HOUR OF POSTOPERATIVE PERIOD TO 24th OF POSTOPERATIVE PERIOD.

VO - VAS SCORE OF 0

V2 - VAS SCORE OF 2

V4 - VAS SCORE OF 4

